



**AGRICULTURAL RESEARCH INSTITUTE**  
**PUSA**







# TROPICAL DISEASES BULLETIN

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1913.



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## ERRATA.

- p. 268. FRANCHINI'S paper, paragraph 5, third line, for "*Maccellie anophelie*" read "*Baccellie anophelie*."
- p. 368, third line from top, for "*Herpetomonas vestimenti*" read "*Herpetomonas pediculi*."
- p. 464, sixteenth line from top, for "cytosome" read "cytostome."
- p. 468, NICOLLE & CONOR'S paper, paragraph 1, sixth line, for "*Ixodus*" read "*Ixodes*."

# TROPICAL DISEASES BUREAU.

## TROPICAL DISEASES BULLETIN

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[No. 1.]

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### TYPHUS.

BURNS (Wm. C.). **A Recent Outbreak of Typhus Fever.**—*Hospital*.  
1913. Feb. 8. Vol. 53. No. 1388. pp. 509-511.

The author describes an outbreak of typhus occurring in Eriskey, a small island of the Outer Hebrides. Thirteen years previously there had been an epidemic of typhus in the island and, among others, all the inmates of one house had perished. The succeeding tenant, fearing to occupy this house, built a new abode, leaving the old one in disuse. The kitchen midden of this old house, a shallow mound chiefly composed of peat ashes, had lain undisturbed during those thirteen years. On this occasion, however, the woman of the new house dug up the old midden and spread the ashes on her potato patch as manure. Within fourteen days she was down with typhus fever and all the other cases were proved "contacts" from this. The kitchen midden was situated on a small mound within a few yards of the seashore, drenched with salt spray during the greater part of the year, and at all times fully exposed to the action of the sun, wind, and rain.

The author remarks that the conditions under which this outbreak occurred show that the virus of typhus is capable of surviving for indefinite periods, in positions and under conditions that would seem entirely to preclude the agency of lice.

There were in all ten cases with three deaths. The fatal cases were in people of 49 years and over; the survivors 31 and under.

[Assuming that this outbreak was correctly diagnosed as typhus, it is certainly difficult to see any manner in which lice could have been responsible for the infection. However, body lice are said to be very much in evidence in some of the houses of this locality, and if any case of typhus had been introduced, there would be ample opportunities for its spread by the agency of these insects. It is certainly almost impossible to conceive that such a fragile virus as that of typhus is capable of surviving for thirteen years in a midden exposed to the conditions described by the author.]

E. Hindle.



**MOLODENKOFF (A.).** **Das Fleckfieber bei Kindern nach dem Material des Morosoffschen Kinderkrankenhauses in Moskau während der Epidemie des Jahres 1911.** [Typhus in Children at the Morosoff Children's Hospital, Moscow, during the Epidemic of 1911.]—*Arch. f. Kinderheilkunde*. 1912. Dec. 10. Vol. 59. Nos. 3-4. pp. 199-233. With 14 curves: also (in Russian) in *Meditsinskoe Obozrainie*. Moscow. 1912. Vol. 77. No. 10. pp. 958-983.

The author has made a very careful study of the occurrence and symptoms of typhus amongst children. During the year 1911, 115 cases of typhus in children of from one to fourteen years of age were observed in the Morosoff Hospital at Moscow. In all the disease ran a very mild course and not one died from it, although the general mortality during this epidemic was about eight per cent. Infants seem to possess a natural immunity against the infection which gradually diminishes as they grow older and the author brings forward both statistical and clinical observations in support of this view. Thus not a single case of typhus was noticed amongst children less than one year old and only seven amongst those of one to three years. Moreover the severity of the attack was directly proportional to the age of the children, the mildest cases occurring among the youngest children and the most severe amongst the elder ones.

The author gives very detailed accounts of the clinical symptoms of the disease in children, for which the reader should refer to the original paper. In conclusion some notes are given on the epidemiology and mode of infection of typhus.

Typhus is a disease of the lowest classes, arising in districts where people are crowded together in the absence of any hygienic conditions; only under these circumstances does it ever assume the proportions of an epidemic and it always disappears under improved surroundings. Although there is little danger of direct infection from typhus patients when the latter are kept under hygienic conditions, any contact with unwashed patients or their clothes or bedding is very dangerous, as shewn by the number of cases occurring amongst washerwomen, porters, hospital attendants, etc. Finally, the author shews how well the epidemiology of typhus agrees with the view that the disease is transmitted entirely by parasitic blood-sucking insects.

E. H.

**CARLAN (Decio).** **El Tifus Exantemático en Madrid.** [Typhus Fever in Madrid.]—*El Siglo Medico*. 1913. Apr. 5. Vol. 60. No. 3095. p. 209.

The author protests against the exaggerations of the Press regarding the present epidemic of typhus fever in Madrid. He points out that the disease is restricted to the poorer quarters of the town and that during the past fifteen days there have only been about 100 cases out of a population of 600,000. Moreover the epidemic is not increasing and there is every hope of it soon disappearing.

E. H.

**LEE (Roger I.). Typhus Fever (Brill's Disease) at the Massachusetts General Hospital in Ten Years (Oct. 1, 1902 to Oct. 1, 1912).—**  
*Boston Med. & Surg. Jl.* 1913. Jan. 23. Vol. 168.  
No. 4. pp. 122-127.

During the past ten years the author has studied all the cases at the Massachusetts General Hospital that had been clinically diagnosed as typhoid fever or cases of fever without definite etiology. As a result it is shown that during this period typhus fever, in a mild and sporadic form, has been present in Boston and its vicinity, occurring in the ratio of about one case of typhus to 47 cases of typhoid fever. In all, 28 cases were observed that corresponded fairly well with BRILL's description of the disease and in all these patients both Widal's reaction and blood cultures gave negative results. The majority of these cases were in persons of Russian nativity, strongly suggesting that the disease was originally imported from Russia. It is interesting to note that, as in BRILL's cases of typhus, the records of the patients do not show any constant infection with lice.

E. H.

**PAULLIN (James Edgar). Typhus Fever with a Report of Cases.—**  
*Southern Med. Jl.* 1913. Jan. Vol. 6. No. 1. pp. 36-43.  
With 5 charts.

In this paper the author describes six cases of typhus occurring in Atlanta, Georgia. Detailed clinical symptoms are given and also the temperature charts of five of the patients.

The symptoms in all cases were typical of rather mild attacks of typhus. Repeated attempts to obtain the Widal reaction with *B. typhosus* and *B. paratyphosus* gave entirely negative results. Blood cultures were made in all except the first two cases, but all remained sterile. None of the patients suffered from any relapses and there were no complications.

[The remarkable mildness of many of the typhus epidemics of America is worthy of notice, and led BRILL to regard it as a separate disease. Subsequent investigations, however, have shown that "Brill's disease" is identical with typhus and the observations of Paullin clearly prove that this disease is endemic in the United States.]

E. H.

**GOLDBERGER (Joseph). Typhus Fever and Typhoid Fever. A Report on Papers read at the Southern Medical Association Meeting at Jacksonville, Fla., November 12-14, 1912.—U.S. Public Health Rep.** 1913. Jan. 10. Vol. 28. No. 2. pp. 63-64.

In this note the author calls attention to PAULLIN's paper (see above) recording the occurrence of typhus fever in Atlanta, Georgia, a record that is of great practical importance as it confirms the view that typhus is endemic in the United States of America.

E. H.

ANDERSON (John F.) & GOLDBERGER (Joseph). **Studies on Immunity and Means of Transmission of Typhus.**—*U.S. Hygienic Laboratory Bull.* No. 86. 1912. Oct. pp. 81-138.

The present bulletin consists of a collection of studies on typhus, most of which have been previously published in various journals. A condensed account of the first part of the section on immunity and means of transmission of typhus (in monkeys) has also been previously published under the title of "Natural and Induced Immunity to Typhus Fever."

In the present article charts are given of many of the cases previously described and for the author's conclusions the reader is referred to the account of the previous paper (see this *Bulletin* Vol. 1, p. 401).

In addition the authors attempted to infect both guinea-pigs and rabbits with typhus. The guinea-pig is susceptible, reacting in essentially the same way to an inoculation with typhus blood as does the monkey. One attack confers immunity from a second. A very small proportion of rabbits may also be susceptible but the results are inconclusive.

A number of experiments were performed with animals immune to other diseases and as a result it is shewn that the immunity conferred by an attack of typhus is specific.

The preventive action of immune serum in monkeys is shewn in a number of experiments. Thus typhus-immune serum obtained between the fifth and fourteenth day of convalescence has protective value when injected simultaneously with, or within 48 hours after, the inoculation of the virus. Immune serum obtained on the thirtieth day of convalescence seems to have no appreciable protective influence. When immune serum and virus are inoculated simultaneously, no immunity results, but when the serum is injected 48 hours after the virus, an immunity, probably active in nature, may perhaps result. It is evident, however, that the therapeutic value of immune serum is at best slight, and at the present time of no practical importance. In addition vaccination with crushed lice suspension, heated to 60° C. for 30 minutes, was attempted with negative results.

The experiments on the transmission of typhus by the body-louse are in continuation of the work of RICKETTS and WILDER and also NICOLLE, COMTE and CONSEIL. The bite of the body-louse may perhaps be infective within four days after the infecting feed. An attempt to inoculate a monkey with typhus by means of freshly hatched body-lice, the offspring of infected parents, gave entirely negative results and therefore satisfactory evidence of the hereditary transmission of the virus in the louse remains to be adduced.

Attempts to transmit the disease by means of the bites and subcutaneous injection of crushed bed-bugs were unsuccessful and these results are in harmony with the epidemiological characters of the disease.

One experiment was performed to test the infectivity of the buccal and pharyngeal secretions in cases of typhus. The

experiment shewed that they were not infective and the authors consider, therefore, that droplet infection plays no part in the transmission of this disease.

E. H.

ANDERSON (John F.). **Some Recent Work on Measles and Typhus Fever.** (Proceedings of the Johns Hopkins Medical Society 1912, Dec. 2.)—*Bull. Johns Hopkins Hospital*. 1913. Apr. Vol. 24. No. 266. pp. 121-124.

The author give a general discussion of certain advances in our knowledge of the communicable diseases, followed by an account of some recent work on measles and typhus fever, especially that done by GOLDBERGER and himself (see this *Bulletin*, Vol. 1, pp. 401-402). The paper contains nothing new.

E. H.

RABINOWITSCH (Markus). **Ueber die Empfänglichkeit der Ferkel für Flecktyphus.** [The Susceptibility of Young Pigs to Typhus.]—*Arch. f. Hygiene*. 1913. Vol. 78. Nos. 4-5. pp. 186-192.

The author briefly mentions that both rabbits and guinea-pigs may be infected with typhus by the injection of either the blood from a typhus patient, or cultures of *Diplobacillus exanthematicus*. In both these animals the symptoms of the disease are very slight and therefore experiments were made in order to find some more susceptible species. With this object two four-day old white pigs were injected with cultures of *Diplobacillus exanthematicus*. The first pig was injected with a one-month old culture and after an incubation period of only four days developed a typical attack of typhus. On the seventh day after the inoculation small petechiae developed on the belly of this animal and during the next two days became very numerous and spread to other parts of the body, so that the whole skin was covered by a typical exanthem. On the sixteenth day the petechiae had very much diminished in number and by the twenty-first had completely disappeared.

The second pig was injected with a three-months old culture. The exanthem was not developed until the fifteenth day after inoculation and was much less intense than in the other pig, and was only present for four days. In both animals the examination of the blood revealed the presence of *Diplobacillus exanthematicus*.

It appears, therefore, that young pigs are very susceptible to typhus, and that the injection of cultures of *Diplobacillus exanthematicus* is followed by the development of the typical exanthematous eruption.

E. H.

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## MALARIA.

HERMS (William B.). **Malaria: Cause and Control.** xi + 163 pp. With 30 text-figs. 8vo. 1913. New York: The Macmillan Company. [6s. 6d. net.]

The author commences his book by discussing the economic loss due to malaria in the State of California. The Board of Health there estimates that the annual loss amounts to \$2,820,400. He gives a list of the *Anopheles* mosquitoes found in California and states that *Anopheles maculipennis* is the most frequent carrier of malaria. A clear and short account is given of the malarial parasite in the human host and in the mosquito. He describes the life history and habits of the mosquito and gives an account of the antimosquito campaigns employed in the several districts in California. He finds that malaria is the principal cause of absences from the rural public schools in the greater part of the malarial infested districts. Three-fourths of the malaria in California is found in nine out of the 24 malarial counties. The book contains useful information regarding the methods of conducting an antimalarial campaign in districts containing an intelligent white population.

D. Thomson.

SOUTHERN MEDICAL ASSOCIATION. **Preliminary Report of the Commission of the Southern Medical Association for the Study and Prevention of Malaria. Year ending November 4. 1912.**—*Southern Med. Jl.* 1913. Apr. Vol. 6. No. 4. pp. 219-226.

The Commission reports the existence of all three kinds of malaria in Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, N. Carolina, Oklahoma, S. Carolina, Tennessee, Texas and Virginia. Benign tertian is most prevalent in the more northern States while malignant tertian is more common in the south. Pernicious malaria is reported in all. So far as can be ascertained from reports of the local practitioners, the number of cases of malaria varies from 25 to 800 per 1,000 of the population, according to the county or state.

*Antimalarial measures:*

1. Apparently no official steps have been taken to make a study of the Anophelines in any of the States.
2. The percentage of houses screened in 15 districts reporting malaria varied from 10 per cent. or less in country areas to 95 per cent. in the larger cities.
3. Drainage and oiling is carried on in one-third to one-half of the infected counties.
4. In 76 per cent. of the infected areas patients suffering from malaria are unscreened and therefore may be frequently a source of infection to mosquitoes.
5. Quinine prophylaxis is resorted to in only 39 of 155 infected counties and in only 3 districts is any attempt made to diagnose and treat latent malaria or malarial carriers.

6. In more than half of the infected counties no public instruction is given.

7. Quinine and calomel is the staple treatment. In about 90 per cent. of the reports, however, it is stated that this treatment is pursued only while the symptoms of malaria are present.

*An estimate of the number of cases, deaths and economic loss from malaria annually in certain of the Southern States.*

*Summary of Estimate.*

State.	Deaths.	Cases.	Total Cost.
			\$
Alabama ....	1,222	122,200	3,666,000
Florida ...	307	30,700	921,000
Georgia ...	1,194	119,400	3,482,000
Kentucky ..	374	37,400	1,122,000
Louisiana ...	160	16,000	480,000
N. Carolina ...	538	53,800	1,614,000
S. Carolina ...	463	46,300	1,389,000
Tennessee ...	629	62,900	1,887,000
Texas ..	362	36,200	1,086,000
Virginia ...	407	40,700	1,221,000
	5,656	565,600	16,868,000

D. T.

VON EZDORF (R. H.). i. **Malarial Fevers in Alabama. A Study of the Prevalence and Geographic Distribution of the Disease throughout the State during the Calendar Year 1912.**—*U.S. Public Health Rep.* 1913. Apr. 4. Vol. 28. No. 14. pp. 641-644. With map.

ii. **Preliminary Studies of Malarial Fevers in Alabama.**—*Southern Med. J.* 1913. Apr. Vol. 6. No. 4. pp. 226-229.

i. The author gives a map of Alabama with its counties showing the distribution of deaths from malaria. The information was obtained by sending 2099 circular postal cards to the physicians in the State, calling for certain information regarding malarial fever. The number of replies received was 431.

The total number of deaths reported as due to malaria in the State for the year 1912 was 546. Of these 233 were in white and 313 in coloured people. 32.6 per cent. of this total of malarial deaths occurred in children in the first decade of life.

ii. The author's studies include statistical, epidemiological and scientific observations. His conclusions are as follows:—

"1. All forms of malarial fevers prevail in the State of Alabama.

"2. The types of infection, in the order of prevalence, are: Tertian, aestivo-autumnal, and quartan.

"3. The colored race is a large factor in the spread of infection, in whom the chronic type of malarial infection is proportionately greater than in the whites.

"4. To obtain the index of infection in a community, a blood examination should be made of adults as well as children of those who are permanent residents of such locality.

"5. Latent or known chronic cases should be treated vigorously for at least one month, and then with prophylactic doses of quinine for the period from April to November.

"6. Prophylactic doses of quinine should be taken by all persons in Alabama living in the localities known to be infected, between the period of April 1 to November 1. The prophylactic dose advised and used by me is 10 grains of quinine sulphate, given in 5-grain doses morning and evening, every Saturday and Sunday of the week.

"7. The microscope should be more generally used for making the diagnosis or confirming the diagnosis of malaria, or if practicable, advantage should be taken of the State Laboratory where examinations of blood will be made."

D. T.

**KAUFMANN (J. B.).** Account of an Outbreak of Malaria on the U.S.S. "Tacoma" resultant upon a Visit to Tampico, Mexico.—*U.S. Naval Med. Bull.* 1913. Apr. Vol. 7. No. 2. pp. 301-302.

This outbreak of malaria on board the U.S.S. "Tacoma" comprised 96 cases, 7 being readmissions; 39 were benign tertian and 57 malignant tertian. The first of the cases appeared 15 days after the arrival of the vessel in Tampico. It is interesting to note that this outbreak occurred despite the fact that the sailors were given 10 grains of quinine daily as a prophylactic from the day the vessel arrived in that port. Later the amount of quinine was reduced to 5 grains daily and 10 grains once a week. Mosquito nets were used at night but the men were bitten severely by mosquitoes in the afternoon while at work. A total of 333 days of sickness was caused by this outbreak. The vessel remained only 17 days in Tampico.

D. T.

**BEACH (T. B.) & LEESON (H. H.).** An Outbreak of Malaria in "F" Company, 2nd Devon Regiment.—*Jl. R. Army Med. Corps.* 1913. Apr. Vol. 20. No. 4. pp. 450-452. With 1 chart.

The authors describe an outbreak of 17 cases of malaria in "F" Company, 2nd Devon Regiment. The disease had been contracted in Cyprus at a place called Zeegoe where the men were badly bitten by mosquitoes. Next day they embarked for Alexandria. The fever outbreak commenced about 12 days later. Quartan parasites were found in three of the cases, and malignant tertian ring forms in two cases. The disease responded readily to quinine treatment.

D. T.

#### EXPERIMENTAL.

**RIEUX (J.).** Mode d'Action de la Quinine sur *Plasmodium vivax* (var. *magna* du Prof. Laveran) de la Tierce et de la Double-Tierce bénigne de Rechute.—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 153-156.

The author observed the effect of quinine in a number of cases of benign tertian malaria, some of which were double tertian and some relapses. Basic hydrochlorate of quinine was used in all the experiments in doses of 0.25 grammes four times daily at 6 a.m., noon, 6 p.m. and midnight. The blood smears were

fixed for 10 minutes in absolute ethyl alcohol and stained with Giemsa, 1 in 10, for 10 minutes. No cover glasses or Canada balsam were used. The technique in all the experiments was identical. His observations were as follows:—

(1) When the quinine administration was commenced during the febrile attack, i.e. when the schizonts were very young, it was noticed that their development became arrested, some showed no increase in size after 24 hours, and those which did grow showed true degenerative changes indicated by difficulty in staining of the protoplasm, which remained sometimes almost invisible. The nuclear chromatin persisted sometimes in the form of a single granule, sometimes it was polymorphic in character with various shapes. The clear area around the chromatin disappeared. The infected corpuscles showed occasionally a marked total or central decolorisation. Those young schizonts which were arrested in their development and degenerated disappeared totally from the peripheral blood in 48 hours from the commencement of the treatment.

(2) If the quinine administration was commenced the day after the access of fever when the schizonts were therefore half grown, they also had their further development arrested, but this did not occur in all of them, some being able to continue their growth to sporulation. Those which were killed showed the same signs of degeneration described, viz.: inability of the protoplasm to take up the stain (sometimes it was broken up into two or four portions), fragmentation and irregularity in the shape of the chromatin, and decolorisation of the containing corpuscles. The Schüffner's granules also seemed to be finer and less numerous than normally. These degenerated parasites disappeared later from the blood, without one being able to detect the details of their disappearance.

(3) When the quinine was given in the above doses at the time of the development of sporulating forms, it was noticed that they showed no appreciable degenerative changes and sporulated normally, causing an attack of fever.

(4) The action of the quinine on the gametes was less rapid, but it caused in them also a degeneration similar to that in the schizonts and they finally disappeared under the prolonged action of the drug.

D. T.

THOMSON (John Gordon) & THOMSON (David). **The Cultivation of one Generation of Benign Tertian Malarial Parasites (*Plasmodium vivax*) in Vitro, by Bass's Method.**—*Ann. Trop. Med. & Parasit.* 1913. Mar. Vol. 7. No. 1. pp. 153-164. With a plate by H. B. FANTHAM, and 1 chart.

The authors succeeded in cultivating the benign tertian malarial parasite on two occasions. They draw the following conclusions from their cultivation experiments regarding the benign tertian and malignant tertian parasites.

"(1) The benign tertian malarial parasite is capable of being cultivated up to the stage of sporulation, for at least one generation.

"(2) In our cultures the growth of the parasite from young rings to sporulating forms took place more rapidly than in the blood of the patient.



"(3) The cultures of benign tertian differed from those of malignant tertian in that there was no tendency to clumping of the parasites in the former, either before or during sporulation.

"(4) This difference appears to us to explain in a satisfactory manner why only young forms of malignant tertian are found in the peripheral blood, as the clumping tendency of the larger forms causes them to be arrested in the finer capillaries of the internal organs. It also explains the tendency to pernicious symptoms, such as coma, in malignant tertian malaria. All stages of the benign tertian parasite are found in the peripheral blood, and there are seldom pernicious symptoms, because there is no tendency to clumping.

"(5) Heavily pigmented, compact parasites were found in our cultures of benign tertian on the fifth day. On the eighth day these had grown larger, and their appearance suggested the development of gametocytes.

"(6) The malignant tertian parasite (*P. falciparum*) is capable of producing, in maximum segmentation, thirty-two spores. On the other hand, benign tertian (*P. vivax*) produces, as a rule, during maximum segmentation, sixteen spores; sometimes more may be produced, but the number is never thirty-two.

"(7) The pigment in *P. falciparum* collects into a definite, circular, and very compact mass early in the growth of the parasite. On the other hand, during the growth of *P. vivax* the pigment remains scattered in definite granules throughout the body of the parasite, till just before segmentation, when it collects into a loose mass of granules in the centre of the full-grown *Plasmodium*."

D. T.

CANTIERI (Collatino). **Le Sieroreazioni per il Tifo (Vidal), paratifi, Bacterium coli e Melitense (Wright) col Siero di Sangue Malarico.** [The Sero-Reaction for Typhoid, Paratyphoid, *Bacillus coli* and *Micrococcus melitensis* with Malarial Blood Serum.]—*Riv. Crit. di Clin. Med.* 1913. Mar. 15. Vol. 14. No. 11. pp. 161-168.

The author states that MENNELLA found a positive Widal reaction with malarial blood serum. STEFANELLI found the Wright reaction positive in ten per cent. of various infections including some cases of malaria. BIRT and LAMB however found this reaction always negative in twenty cases of malaria, while GARDEN found it always negative in 122 cases of typhoid, malaria, tuberculosis, etc. In his own experiments the author used an emulsion of *Micrococcus melitensis* made from an 8 to 24 hours' growth on agar. The other germ emulsions were made from 24 hours' cultures in broth media. In 58 malaria cases (17 primary infections; 32 relapses; undetermined 3, and malarial cachexia 6) these sero-diagnostic tests under the most varied conditions were negative.

D. T.

#### TREATMENT.

REED (E. U.). **A Case of Malaria treated with Salvarsan.**—*U.S. Naval Med. Bull.* 1913. Apr. Vol. 7. No. 2. pp. 255-256.

The author describes a case of benign tertian malaria in a seaman. This patient had three severe relapses although he had received two months treatment with large doses of quinine sulphate after the first two. [The exact dosage is not given.] The third relapse was treated by an injection of 0.6 grams of salvarsan into the buttock. The fever abated next day and no further relapse occurred in the year following.

D. T.

## PROPHYLAXIS.

CORVINO (Raffaele). **Lotta Antimalarica in Cancellò ed Arnone.** [Antimalarial Campaign in Cancellò and Arnone, (Province of Caserta, Italy).]—*Propaganda Antimalarica*. 1912. Dec. 31. Vol. 5. No. 6. pp. 136-139.

The locality in question was so infested with malaria during 1895-1900 that in August there was not a single person who was not infected. Owing to the labours of the author circumstances have gradually improved, so that now no case of pernicious malaria has been seen for the last five years. He thinks that this result is due principally to the administration of state quinine.

D. T.

CACACE (Ernesto). **Educazione Antimalarica e Profilassi Antimalarica Scolastica in Italia nel 1911.** [Antimalarial Education and Prophylaxis in the Schools in Italy during 1911.]—*Propaganda Antimalarica*. 1912. Dec. 31. Vol. 5. No. 6. pp. 131-136.

The author shows that the results obtained from antimalarial instruction in the schools, by the workers in the various districts, are very favourable. He again urges the authorities to make this school instruction compulsory in Italy.

D. T.

TIMPANO (Pietro). **Profilassi Antimalarica nelle Scuole Elementari di Bova Marittima nel Biennio 1911-12.** [Antimalarial Prophylaxis in the Elementary Schools of Bova Marittima (Calabria) 1911-12.]—*Propaganda Antimalarica*. 1913. Feb. 28. Vol. 6. No. 1. p. 20.

In 1911, 40 pupils received state quinine [dose not stated] from May 15th to August 31st, and they had no fever. In 1912, 29 boys between eight and twelve years of age, coming from the worst malarial focus, received state quinine during June and July in the afternoons before going home. The success of this medication was complete. The author thinks that the free administration of quinine to school children, combined with simple and clear instructions as to the benefit derived from the quinine, would constitute the best antimalarial measure.

D. T.

SERGI (Antonio). **La Malaria nella Riviera Ionica Reggio—Gerace nel 1911.** [Malaria in the Ionic Riviera between Reggio (Calabria) and Gerace in 1911.]—*Propaganda Antimalarica*. 1913. Feb. 28. Vol. 6. No. 1. pp. 17-19.

The author states that the conditions of life and social hygiene in this area are very bad. His statistics however show a decrease in malaria from 11.34 per cent. of the total population in 1910 to 9.80 per cent. in 1911. In 1910 the maximum number of cases, 811, occurred in August and in 1911 the maximum occurred in

September (1,342 cases). He gives further data showing the number of people in receipt of prophylactic quinine and the number of cases, relapses, and deaths from malaria among these. The amount of quinine consumed in 1910 and 1911 was 80,616 grammes and 83,326 grammes respectively. Anophelines were more or less abundant from June to September.

D. T.

ROSSI (Giacomo). **Sul Bonificazione definitiva della Piana di Fondi e Monte San Biagio.** [On the Definitive Drainage of the Piana di Fondi, etc.].—*Propaganda Antimalarica*. 1913. Feb. 28. Vol. 6. No. 1. pp. 1-17.

This paper is a dissertation on the possible difficulties which might arise in an attempt to drain the Piana di Fondi (Caserta, Italy) and Monte San Biagio. The author concludes that there should be practically no difficulty in making the lagoon permanently dry, and expresses the hope that the work, which has been tentatively taken up by many, will now finally be accomplished.

D. T.

BRÜNN (W.) & GOLDBERG. **Das Cisternenproblem bei der Bekämpfung der Malaria in Jerusalem.** [The Cistern Problem in Antimalarial Measures in Jerusalem.].—*Berlin. Klin. Wochenschr.* 1913. Apr. 7. Vol. 50. No. 14. pp. 639-640.

The authors made over 2,000 blood examinations in the Jewish quarters of Jerusalem and found that 21.5 per cent. of these showed malaria. Of 600 people 57 per cent. had splenic enlargement. They believe that badly constructed cisterns must be regarded as the main cause of this malaria, as they are a constant source of breeding of anopheline mosquitoes. The total abolition of these cisterns is not practicable, but the authors have substituted pumps in place of the bucket system, have closed all unnecessary openings and have designed effectual screening for those openings which are indispensable.

D. T.

FRÉES (Joao A. G.). **Un Caso de Aphemia Transitoria Palustre. (Aphasia Motora Verbal, Funcional, de Origem Paludica.)** [A Case of Transitory Functional Motor Aphasia due to Malaria.].—Reprint from *Brazil-Medico*. 1912. [41 pp. Rio de Janeiro: Lyp. Bernard Frères.]

In this paper, read before the Bahia Medical Faculty, the author reports the following case in a native of Bahia, aged 34. The patient suffered from malignant tertian malaria and had numerous crescents in his peripheral blood. He was an alcoholic and syphilis was strongly suspected. He developed a complete verbal motor aphasia without agraphia. This aphasia was transitory and the author concludes that it was malarial in origin from microscopical examination of the blood and from the fact that it completely disappeared under quinine treatment.

He thinks that the aphasia in this case may have been due to parasitic thrombi in the speech centre of Broca, since it is well known that the malignant tertian parasites tend to accumulate and sporulate in the capillaries of the inner organs. The alcoholism and syphilis (suspected) may have further aggravated the tendency to capillary thrombosis due to the malarial parasites.

D. T.

FAZZARI (G. B.). **Malaria ed Epatite Suppurata.** [Malaria and Suppurative Hepatitis.]—*Malaria e Malat. d. Paesi Caldi.* 1913. Mar. Vol. 4. No. 2. pp. 77-79.

The author reports two cases of malignant tertian malaria with suppurative hepatitis. He aspirated 200 cc. of pus in each case. One died. There were no signs of dysentery or hydatids. He believes that the malaria was indirectly the cause of the suppuration by producing changes in the structure of the hepatic parenchyma, rendering the organ more susceptible to infection from suppurative germs, possibly *B. coli* or "*Streptococcus duodenalis*." These germs increase in virulence owing to the devitalised condition of the intestine and other organs. They infect the bile passages or follow the course of the portal vein and are carried by the blood stream to the liver. [Were entamoebae excluded?]

D. T.

WILSON (F. E.). **Note on Three Cases of Splenic Abscess occurring in so-called "Malarial Cachexia."**—*Lancet.* 1913. Apr. 26. p. 1162.

The author states that he has been impressed with the frequency of splenic abscess occurring in cases of so-called malarial cachexia in Persia and by the disappearance of the cachexia on treatment directed against the local condition. He diagnoses such cases from the continuance of fever in spite of thorough quinine treatment, from local evidence of softening, pain or adherence of the spleen to the abdominal parietes, and from the existence of a leucocytosis. He operated on the three cases described; two benefited very markedly and one died. In one case  $1\frac{1}{2}$  pints of pus were evacuated, in the other  $1\frac{1}{4}$  pints. Bacteria were found in the pus but no malarial parasites. [Cases of splenic abscess due to entamoebae have been described. Were they looked for in this case?]

D. T.

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## YELLOW FEVER.

SEIDELIN (Harald). *The Nature and Control of Yellow Fever.*—*Yellow Fever Bureau Bull.* 1913. Jan. Vol. 2. No. 3. pp. 255-271.

This is a paper which was read at the XVth International Congress on Hygiene and Demography at Washington in September, 1912, and consequently it contains much that has appeared since in the author's reports upon his yellow fever expeditions. In reference to the transmission of the disease the author states that "the axiom that yellow fever is transmitted by no other mosquito than *S. fasciata* is an assertion entirely without proof" and again "that yellow fever is transmitted in nature by no other means than by mosquitoes has never been proved, but is extremely probable in view of our knowledge of protozoal diseases." The paper commences with the statement that "Yellow fever is an infectious disease, produced by *Paraplasma flavigenum* and transmitted by *S. fasciata*" (but see this *Bulletin*. Vol. 1. pp. 246-249.

The author considers that the view that a period of twelve days incubation is necessary in the mosquito before it becomes infective "is likely to be correct but cannot be considered exclusively proved," and further that the view that yellow fever blood is infectious during the first three days of the disease only rests upon "very scanty evidence." The responsibility for this dogma, the author says, rests with MARCHOUX and SIMOND but "never has a dogma in medicine been established on evidence poorer than this." With the view that yellow fever is only transmitted at night he seems to agree and points out that MARCHOUX and SIMOND showed that *Stegomyia fasciata* feeds in the day time only at its first feed and takes its subsequent feeds at night time. Hereditary transmission in mosquitoes and infection of mosquitoes by feeding on sugar-water containing triturated, infected mosquitoes, have been observed by MARCHOUX and SIMOND in one experiment of each kind.

After giving a description of *Paraplasma flavigenum*, which the author considers to be the causative parasite of yellow fever, he goes on to explain that though the discovery of the mosquito transmission of the disease promised a speedy extermination of the outbreaks, this expectation has only been realised in such places as Habana, New Orleans, Panama Canal Zone, Rio de Janeiro and Vera Cruz, where powerful governments have been able to insist on prophylactic measures. Two most important points in the epidemiology of yellow fever are the existence of unrecognised and unrecognisable cases. It is asserted that the susceptibility of the individual diminishes with the length of time during which he has resided in a yellow fever infected country. This phenomenon cannot be explained unless it is admitted "that the (relative or absolute) immunity has developed as a consequence of acquired infection." This explanation is clear when applied to foreigners, and MARCHOUX and SIMOND, OTTO, BOYCE and others have extended the idea to explain the relative immunity of the native. Infection takes place in childhood, producing nearly always an atypical form of the disease. The child growing up is repeatedly bitten by infected *Stegomyia* which either

produce no disease or only a mild febrile disorder. Under such conditions many individuals act as microbe carriers and serve as the starting point of an epidemic when, for example, a large number of non-immunes are suddenly introduced to the district. Another factor which may influence the native susceptibility is, in the author's opinion, a certain degree of hereditary immunity, antibodies having passed from mother to child.

The author gives the following directions for the demonstration of his parasite in the peripheral blood of cases of the disease.—

"The demonstration of *P. flavigenum* is difficult; a very thorough examination of well prepared and intensely stained blood smears is necessary, and the best microscopic outfit is none too good. It is almost useless to work with an ordinary 1/12" immersion lens; truly, I have often seen the parasites with such a lens, when I was aware of their existence and had localized them before, but for the detection of *Paraplasma* I consider an apochromatic immersion lens and compensating oculars a necessity. The examination should be repeated, if a negative result is obtained, and the case continues suspicious. My belief is that this means of diagnosis will become as essential in yellow fever as is the bacteriological examination in plague and cholera."

Under the heading of prophylactic measures the author considers the protection of mosquitoes against acquiring infection from human individuals and the protection of non-immunes against the bites of infected mosquitoes. This entails the destruction of adult mosquitoes, the prevention of their breeding, the early recognition and isolation of cases of the disease. The author then gives his views as to the measures which should be put into force in three illustrative cases, viz.:—Endemically infected places; exposed places; epidemic outbreaks. In the first case as indeed in all three, complete mosquito destruction is the ideal to be aimed at. In the second case anti-mosquito quarantine, consisting in the fumigation of all ships arriving from infected or suspected ports, must be undertaken unless it be shown by certificate that during its stay a safe distance of 200-500 metres from shore had been kept and that loading and unloading had been effected by means of mosquito-proof lighters. As regards passengers they should undergo an initial examination and sign a declaration that they will report to the health officer twice daily for two weeks. In event of any suspicious sign they could be isolated. This observation of passengers is, in the author's opinion, preferable to the real quarantine system. In the case of epidemic outbreaks the immediate undertaking must be the destruction of the adult mosquitoes together with the detection and isolation of the cases.

C. M. Wenyon.

WHITE (Joseph H.). *The Dissemination and Prevention of Yellow Fever*.—*Amer. Jl. of the Med. Sciences*. 1913. Mar. Vol. 145. No. 3. [No. 492.] pp. 378-386.

The author points out that, though it has been demonstrated that yellow fever is transmitted by *Stegomyia calopus*, some still cling to the idea that the disease can be conveyed by fomites. The author shows how untenable this view is by reference to his hospital experience with yellow fever cases which were completely screened from mosquitoes without any precautions being taken against other possible means of spread of the infection.

He describes the habits of the *Stegomyia calopus* and points out that it is a domesticated mosquito which, when once it has taken up its abode in an inhabited room or house, seldom leaves it. The author is convinced that to become infected with yellow fever "one must enter a room, with a dim light, where yellow fever has been at least fifteen days before." Under certain exceptional circumstances the *Stegomyia* will leave the sick room but only when this is completely abandoned and left without water. With few exceptions the only breeding places of the *Stegomyia* are those receptacles, cisterns, containers, etc., which are the direct result of human habitation; but though this is the case, in yellow fever prophylaxis it is well to attack all mosquitoes alike, for it is just possible that the *Stegomyia* will be driven to breed elsewhere if it finds its breeding places of selection destroyed. In the case of fountains and similar collections of water the introduction of small fish is recommended. Roof gutters are a prolific source of danger, so that the ideal way is to dispense with these structures and allow the water to fall from the roof on to paved ground and thence flow away. In dealing with endemic centres, where naturally the measures adopted will extend over a considerable period of time, it is hardly possible or advisable to attack the adult mosquitoes themselves: but in an epidemic this is very important and one cannot too carefully isolate the sick man and his accompanying mosquitoes, which latter are to be destroyed by fumigation when the patient is removed to another house after the third day of his illness. Neighbouring houses if connected are to be treated also, but the author deems it unnecessary to fumigate neighbouring detached houses. A reference is made to the author's work in connection with the extermination of yellow fever in New Orleans which was commenced in August 1905. By the end of October there was a total elimination of yellow fever, nearly all *Stegomyia* and most of the *Anophelines* and *Culicidae*, and this in 44 square miles of territory with a population of 335,000 inhabitants who had presented to that time 600 cases of the disease.

C. M. W.

**BOUET & ROUBAUD. Mission de la Prophylaxie de la Fièvre Jaune au Sénégal. Instructions relatives à la Lutte Antilarvaire.—**  
*Ann. d'Hyg. et Méd. Colon.* 1912. Oct.-Nov.-Dec. Vol. 15.  
 No. 4. pp. 742-750.

In this paper the authors write that in an inspection of the various centres in Senegal, especially in the provinces of Cayor, Baol and Saloum where the Sanitary Service has been conducting antilarval measures, they have noted that in spite of all efforts the measures adopted have resulted in no decided improvement. They indicate that this failure is not due to any negligence on the part of those in charge of affairs but rather to a lack of knowledge of the mosquitoes and their habits.

The authors then review the best methods for finding the larvae of the various mosquitoes and the plan they have found most convenient and simple for carrying on the work of the hygiene brigade. As regards the larvae it is pointed out that these vary with the habits of the mosquitoes themselves, which are considered under the following headings:—(1) Mosquitoes of

pools; (2) Mosquitoes of large swamps and lagoons; (3) Mosquitoes of crabs' burrows; (4) Mosquitoes of domestic receptacles.

Under the first heading are considered the Anophelines, for they breed in ponds of stagnant water in which algae grow and on the borders of which there is vegetation. In the second place the mosquitoes which frequent the large expanses of water, the margins of lagoons, deltas of rivers, are chiefly *Mansonia*. It is fortunate that these mosquitoes do not transmit either malaria or yellow fever, for they can be exterminated only by extensive irrigation works. Thirdly, certain mosquitoes breed in the small holes made by the terrestrial crabs which live near the sea or collections of brackish water. These can only be dealt with by petrolising each hole individually. Lastly the mosquitoes of domestic receptacles are the *Stegomyia* which are to be kept under by constant examination. A long list of the various situations in which these mosquitoes breed is added and it is pointed out that the mere emptying of an infested receptacle is not sufficient unless measures are taken to prevent water collecting again in a short time; for the eggs and larvae may survive if complete drying has not taken place. A section is devoted to the equipment and duties of an antimosquito brigade and the kind of inspection to be carried out in order to render effective the measures adopted.

C. M. W.

- i. AUGÉ. *Une Observation de Fièvre Jaune au Dahomey*. [Clinique d'Outre-Mer.]—*Ann. d'Hyg. et Méd. Colon.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 181-184.
- ii. *Observations de Fièvre Jaune au Sénégal*. [MARQUE.]—*Ibid.* pp. 184-193.

The first note contains the description of a typical case of yellow fever which terminated fatally.

The second note describes three cases of the disease, two of which were fatal.

C. M. W.

**WEST AFRICA. Report on Certain Outbreaks of Yellow Fever in 1910 and 1911.—1913.** 108 pp. With 6 maps. London: Printed by Waterlow & Sons.

This interesting report was compiled by Drs. A. E. HORN and T. F. G. MAYER and is based on various reports received from the Colonies on the West Coast of Africa. It is full of matter of the utmost importance to those who will be called upon to combat yellow fever on the West Coast and to do justice to it in a short space is impossible. Details of 64 cases are given together with a minute account of the local conditions associated with their origin and the prophylactic measures adopted to check the spread of the disease. On pages 21 and 22 is to be found a tabular statement of the cases occurring in the years 1910 and 1911 and this is reproduced here.

There are spot maps of Freetown, Seccondree, Accra and Bathurst, showing where the cases occurred and two maps of West Africa showing (a) the epidemics of 1910 and 1911, and (b) all recorded epidemics and the distribution of *Stegomyia fasciata*.



TABULAR STATEMENT OF THE CASES OF YELLOW FEVER ON THE WEST COAST OF AFRICA.

Place.	Case No.	Race.	Age.	Occupation.	Date.	Result.	Quinine Prophylaxis.	Blood examination for Malaria Parasites.	Remarks.
Freetown	1	Syrian ...	20	Trader	1910. April 7-17	Death ...	Probably No	Malignant Tertian	Malaria followed by Yellow Fever.
	2	Syrian ...	—	Trader	April 17	Death ...	— No	—	No details of illness.
	3	Syrian ...	23	Trader	May 4-5	Death ...	Probably No	Negative.	No details of illness.
	4	Syrian ...	15	Trader	May 5 ...	Death ...	—	—	
	5	Syrian ...	42	Agent	May 6-7	Death ...	Probably No	Negative.	
	6	European ...	—	Trader	May 9-14	Death ...	—	—	
	7	Syrian ...	25	Trader	May 13	Death ...	Yes ...	Negative	No details of illness.
	8	Syrian ...	50	Trader	May 18-25	Death ...	—	Negative	Travelling in Protectorate before attack.
	9	European ...	60	Trader	May 26-31	Death ...	—	Negative	Ten years in West Africa.
	10	European ...	28	Govt. Official	June 5-8	Death ...	Yes, regularly	Not made	An ambulant case.
	11	European ...	30	Soldier	June 25-July 13	Recovery	Yes, regularly	Negative	Taken ill at Yonni in the Moyamba district; an ambulant case.
	12	Syrian ...	35	Trader	July 13-17	Death ..	Probably No	Negative	An attack of Malaria preceded the attack of Yellow Fever by seven days.
	13	Syrian ...	—	—	July 18-22	Death ...	—	Negative.	The post-mortem, of which there are no records, was thought to confirm a diagnosis of Yellow Fever.
	14	Negro ...	23	Clerk	July 30-Aug. 2	Death ...	No ...	Not made.	
	15	Negro ...	24	Labourer	Aug. 1-5	Recovery	No ...	—	
	16	European ...	25	Trader	Aug. 1-17	Recovery	Yes, regularly	Negative	An ambulant case.
	17	European ...	27	Govt. Official	Aug. 11-20	Recovery	Yes, regularly	—	
	18	Negro ...	34	Cook	Aug. 18-27	Recovery	No ...	—	
	19	Negro ...	26	Labourer	Aug. 28-29	Death ...	No ...	—	
	20	European ...	26	Clerk	Sept. 15-22	Death ...	Irregularly ...	Doubtful	History of $C_3H_6O$ ; ambulant case.
Secondree	21	European ...	39	Govt. Official	March 19-24	Death ...	Irregularly ...	—	
	22	European ...	30	Agent's wife	April 12-28	Recovery	Not known ...	—	
	23	European ...	42	Govt. Official	April 20-30	Death ...	Not known ...	—	
	24	European ...	26	Missionary	May 8-10	Death ...	—	—	
	25	European ...	25	Contractor	May 9-13	Death ...	Not known ...	—	
	26	European ...	45	—	May 9-12	Death ...	No ...	—	
	27	Negro ...	—	Labourer	May 16	Death ...	Not known ...	—	
	28	Negro ...	—	Contractor	May 18-21	Death ...	Not known ...	—	
	29	European ...	35	—	—	Death ...	—	—	
	30	European ...	—	—	—	Death ...	—	—	

An attack of Urticaria preceded the Yellow Fever.  
Not a native of Secondree.  
Ambulant case.



**RICE (Thomas E.). Evidence of the Endemicity of Yellow Fever in the Gold Coast Colony.—*Yellow Fever Bureau Bull.* 1913. Jan. Vol. 2. No. 3. pp. 272-274.**

The author reminds his readers that in 1910 he wrote that he considered yellow fever to be endemic amongst the native population of West Africa. He is still of this opinion and considers that no other hypothesis can explain the outbreaks. During the period March 1910-August 1912, 23 cases of the disease were reported amongst the non-immune European population of the Gold Coast and of these 22 were fatal. The logical deduction from this high death rate is that many mild cases must escape recognition. The small number of cases occurring at each outbreak is frequently raised as an argument against the existence of yellow fever in West Africa. This is explained by the fact that the bulk of the population consists of immune natives and that non-immune Europeans live in bungalows usually widely separated and not in crowded streets as they did in the historic epidemic in New Orleans. Yellow fever in West Africa is apt to be a bungalow or house disease rather than one attacking whole streets or districts.

In conclusion the author submits that for the following reasons yellow fever must be regarded as endemic on the Gold Coast.—

"(1) During the 1910 outbreak in Secondee, fifty Europeans and 1,500 natives were resident in the infected area of Commerical Town, Secondee.

Ten Europeans contracted the disease and nine died of it; not a single native died or showed symptoms of yellow fever. Therefore, presumably, the disease was endemic and the natives immune.

"(2) The European who was infected at Saw Mills Camp in 1910 had not been away from his station for three weeks. No cases were observed amongst the natives.

"(3) The European who died at Avibro in 1911 had not left his camp for a month. No cases occurred amongst the natives.

"(4) During the outbreaks at Accra during 1911 and 1912, eight Europeans contracted yellow fever, in each case with a fatal result. Whereas, during the same period ten natives were diagnosed with the disease in a mild form, and all recovered, a fact which points to their having acquired a partial immunity from previous infections."

C. M. W.

**LICEAGA (Eduardo). Annual Report on the Condition of Yellow Fever throughout the Mexican Republic.—*Amer. Jl. Public Health.* 1913. Mar. Vol. 3. No. 3. pp. 263-264.**

The period reported upon extends from November 25, 1911 to August 31, 1912.

There were sixteen cases of the disease with six deaths in Merida. The last case was seen on May 1st. Cases occurred at San Juan Bautista to the number of fifty with 22 deaths between May 1st and August 19th. A battalion of troops proceeding from San Juan Bautista to Frontera was isolated and two cases occurred during the isolation. The author remarks that if the circumstances prevailing have permitted the development of the two foci named it is to be expected that with the new plan of

campaign which is being put into practice the disease will be finally extinguished.

C. M. W.

GARCIA (Pedro). **Notas de Clinica Hematológica. La Nueva Etiologia de la Fiebre Amarilla. Epidemia del Yucatan.**—*Semana Medica.* 1913. Vol. 20. No. 2. pp. 61-67.

This paper is a summary of the work carried out by Harald SEIDELIN on his recent expedition to Yucatan on the subject of yellow fever (see this *Bulletin* Vol. 1, p. 246).

C. M. W.

JACOBI (Carlos). **Un Nuevo Método Profiláctico de la Fiebre Amarilla.** [A New Method of Prophylaxis for Yellow Fever.]—*Annales de la Acad. de Ciencias de la Habana.* 1912. Oct. Vol. 49. pp. 371-384.

This is a reprint of a paper read in 1864. The author points out that yellow fever is generally a benign disease in the months of January and February and is more serious from June to September. Accordingly he suggests that if it can be shown that yellow fever is transmissible by inoculation it might be advisable to inoculate people from benign cases in order to protect them from the possibility of acquiring the more serious type of disease.

C. M. W.

NOC (F.). **Le Diagnostic Biologique des Fièvres Amariles et des Émittentes Biliieuses d'Origine Paludéenne.** [Clinique d'Outre-Mer.]—*Ann. d'Hyg. et Méd. Colon.* 1912. Oct.-Nov.-Dec. Vol. 15. No. 4. pp. 875-879.

Now that yellow fever has been checked by the systematic destruction of *Stegomyia* larvae it often becomes a matter of extreme difficulty to arrive at a diagnosis between bilious remittent fever of malarial origin and the bilious fevers of a mild form (*inflammatoires des Antilles*) which are really yellow fever. This is especially the case in the Antilles and Guiana. The necessity of recognising these mild cases of yellow fever is obvious, for it is such cases which cause the disease to survive through periods when the sanitary condition is excellent. To arrive at a diagnosis three points must receive special attention: (1) Repeated examinations of the blood; (2) Examination of the urine for albumin; and (3) A study of the leucocytic formula. The author mentions the case of a man who suffered from remittent fever, bilious vomiting, jaundice, icterus, and profuse sweating. There was absence of albumin in the urine, slight mononuclear increase, but no malarial parasites in the blood. The patient had taken quinine. Such a case, occurring in a district in which the mild form of yellow fever was endemic, offered great difficulties of diagnosis, which was eventually

arrived at by stopping the quinine and the finding of ring malarial parasites in the blood after a recurrence of the fever and bilious vomiting.

Notes of several cases of fever are given to show how the blood picture may vary in the mild forms of yellow fever. Two periods occur, one of polynuclear increase with diminution or total disappearance of the eosinophiles and another and later in which a general mononuclear increase takes place. The first period is before the remission, the second from the third to the sixth day. Attention to the three points mentioned above will ensure a correct diagnosis.

C. M. W.

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## PAPPATACI FEVER.

NICLOT. **A propos de la Fièvre à Pappataci.**—*Bull. Soc. Path. Exot.* 1912. Dec. Vol. 5. No. 10. pp. 780-781.

A survey of French investigations is made. MIORCEC and LAPLANCHE\* confirmed BIRT's observations on the existence of this malady in Crete; more than half of the French detachment, which numbered 200 men, were attacked in July and August. NICLOT† stated that he had received many specimens of *P. papatasii* from the various military posts in the Oran Division of Algeria during the years 1904 to 1907. In the year 1908 FOLEY and YVERNAULT‡ reported that they had captured these flies at Beni-Ounif in south western Algeria, and SERGEANT found them at Biskra. In France the localities where the phlebotomus has been taken, are according to ROUYER.§ Alpes-Maritimes, Basses and Hautes-Alpes, Isere, the valley of the Oisans, Mount Genève, and the district round Briançon. LEGER and SÉGUINAUD announced their capture in Corsica (see this *Bulletin*, Vol. 1. p. 238).

C. Birt.

- i. FRANCA (Carlos). *Phlebotomus papatasii* (Scopoli) et Fièvre à Pappataci au Portugal.—*Bull. Soc. Path. Exot.* 1913. Feb. Vol. 6. No. 2. pp. 123-124.
- ii. DOS SANTOS (L. Pereira). *Contribuição para o Estudo da "Febre de Papatasii" em Portugal.*—*Medicina Contemporanea.* 1913. Vol. 31. No. 3. pp. 20-23.

i. *P. papatasii* appears at Collares on the coast north of Lisbon in the months August, September, and October. Pappataci fever occurs. The depression during convalescence is sometimes great.

ii. The author gives a short account of the symptoms of the fever and the mode of its transmission for the benefit of his fellow practitioners in Portugal.

C. B.

CASTRO (A.). *Sulla Febbre dei Tre Giorni a Milazzo.*—*Malaria e Malat. d. Paesi. Cald.* 1913. Mar. Vol. 4. No. 2. pp. 68-73.

The author has observed epidemics of three-day fever every summer since 1909 among the troops stationed at Milazzo in the north of Sicily. Last year the outbreak began at the end of June and reached its maximum in July and August, when the admissions rose to 10-30 a day. The usual symptoms of sandfly

\* *Bull. Bi-mensuel Soc. de Méd. Milit. Française.* 1911. Feb. 2. Vol. 5 No. 3. pp. 128-132.

† *Ibid.*, 1911. Mar. 16. Vol. 5. No. 6. p. 193.

‡ *Arch. de Méd. et de Pharm. Milit.* 1908. May.

§ *Bull. Bi-mensuel Soc. de Méd. Milit. Française.* 1911. Mar. 16. Vol. 5 No. 6. pp. 194-195.

fever were noted—severe pains in the head, back, and limbs, discomfort in the epigastrium; pyrexia for three days generally, and considerable debility for four or five days afterwards.

C. B.

SARLO-BISOGNI (Fr.). *Di un'Epidemia di "Febbre dei Tre Giorni" a Francica (Provincia di Catanzaro).—Malaria e Malat. d. Paesi Caldi.* 1913. Mar. Vol. 4. No. 2. pp. 82-84.

A three-day fever which resembled sandfly fever prevailed at Francica in the south of Italy during July and August 1912. During the incubation period of some of the attacks, malaise and a tendency to headache were noted. Convalescence was protracted in some instances to 15 days through lassitude and weakness. Pyramidon was useful in alleviating the symptoms; aspirin, Dover's powder, and salicylate of soda were also employed with benefit.

C. B.

ARAVANDINOS (Anast.). *Klinische Erwägungen über das Dreitage-fieber und die Dengue in Griechenland.* [Clinical Observations on Three-day Fever and Dengue in Greece.] — *Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Feb. Vol. 17. No. 4. pp. 109-120.

In May 1911 the author reported the occurrence of pappataci fever in Athens. Phlebotomi are common in Greece, and though the inhabitants do not suffer to a great extent from sandfly fever, probably because they acquired immunity by being attacked in infancy, yet visitors from other countries usually contract the infection during their first summer of residence. Two English ladies were attacked with this fever in July, two months after their arrival at Athens. The typical symptoms were observed, including leucopenia and slow pulse; phlebotomi were captured in their rooms.

Dengue breaks out in epidemic form every few years at ports along the coast of Greece, when a large proportion of the population is affected. In the Piraeus outbreak, which began at the end of August 1910, the characteristic eruption accompanied with itching and succeeded by desquamation, was rarely wanting; arthritic pains were severe; and in a few instances nephritis, orchitis, and parotitis were sequelae. The distinction between a dengue and a pappataci fever epidemic is unmistakable.

C. B.

NAVY (HEALTH). *Statistical Report of the Health of the Navy for the Year 1911.—1912.* London: Published by H.M. Stationery Office. [Phlebotomus Fever, pp. 53 and 67.]

Under the heading "Pyrexia" in the "Remarks on the Returns of the Mediterranean Station," one hundred and forty-four cases are recorded. A marked resemblance is to be noted

between the cases returned under pyrexia and phlebotomus fever. They chiefly occurred during the hot weather, and almost all were of short duration. Most of them originated at Malta or Platea, but many arose at the ports of the Riviera. The attacks lasted three or four days and were accompanied with high fever at the onset, furred tongue, head- and backache.

In "Remarks of the returns of the China Station," thirty-two cases of pyrexia are recorded, which were mostly of a very mild type; and caused disablement for a few days only. In none was there any resemblance to "Yangtse River fever."

Twenty-two cases of "pyrexia" are included in the returns of the Atlantic Fleet which was in Mediterranean waters during the year. It is stated that some of these were sandfly fever.

Seventeen similar attacks occurred in the "Pandora" when she was at Zanzibar.

[It is to be hoped that this fever will be given its specific name in future Annual Reports. In the Report for 1909 it is stated that the majority of cases recorded under the heading "pyrexia" in the Mediterranean were probably phlebotomus fever. Again in the Report for 1910, there appears the remark that most of the 98 cases of pyrexia in the Mediterranean seem to have been phlebotomus fever. Eighteen similar febrile attacks were contracted off the Siamese coast, the Solomon Islands, and the Queensland coast. Moreover in this volume there is an excellent paper by Fleet-Surgeon KILROY and Surgeon ADSHEAD on Phlebotomus Fever in Crete. The former officer exposed himself for five nights to the attacks of sandflies on Suda Island where the fever is very prevalent, contracted the disease, and infected a healthy man with blood withdrawn from his veins at the end of the first day of his illness.]

C. B.

TAYLOR (E. C.) & KHAN (Mohammed Hakimullah).—*The Diagnosis of Sand-Fly Fever and its Differentiation from Malaria.*—*Indian Med. Gaz.* 1912. Dec. Vol. 47. No. 12. pp. 475-476.

161 cases of sandfly fever were admitted to the Militia Hospital at Parachinar, an Indian frontier post, about 100 miles west of Peshawar, during the months June to September 1912. The flushed face, suffused conjunctivae, injected fauces, slow pulse, complaint of severe aches and pains, and absence of parasites in the blood were marked in the majority of patients. There was pyrexia for one day in 73, for two days in 55, for three days in 24, and for four days or more, in 9 of those who were attacked. 112 cases of malaria came under observation during the same period, in 78 per cent. of which parasites were found. They were distinguished clinically from the phlebotomus infection, by the patients' complaint of "fever," and not of pains, by the absence of congestion of the eyes and fauces after the hot stage had passed, and by the pulse rate being over a hundred on admission.

C. B.



## PHLEBOTOMUS AND ITS BIONOMICS.

MARETT (P. J.). *The Phlebotomus Flies of the Maltese Islands.*—*Jl. R. Army Med. Corps.* 1913. Feb. Vol. 20. No. 2. pp. 162-171.

Of 106 phlebotomi caught and encaged during the month of September 1911, 38 per cent. were male, and 30 per cent. female, *P. perniciosus*; 22 per cent. were male, and 13 per cent. were female, *P. minutus*. 12 per cent. of these flies died during the first twenty-four hours of captivity; more than half were dead at the end of three days; only two survived ten days in the cage; and this was their limit of life in captivity, the average duration of which was four days. *P. papatasi* were fewer in numbers than the above named. *P. nigerrimus* was scarce. The Maltese *P. papatasi* differs from the Dalmatian variety in the third segment of the antennae of the latter being only one-third longer than the fourth segment; and in the terminal segment of the superior clasper of the male being longer than the inferior clasper. The Austrian variety, however, is found also in Gozo. *P. papatasi* breeds in caves and embankments, *P. perniciosus* and *minutus* in rubble walls and bastions.

A larger number of pupae and larvae were found in the year 1911 than in the previous summer, when the rubble walls of Malta were investigated. In 1911 a cave in the island of Gozo was a special object of study: on more than one occasion two pupae were found on one stone. In hot weather the eggs hatch in six to nine days; if the tubes containing them were somewhat dry, this was delayed until twenty days. The larval stage lasts about eight weeks in summer, but under the influence of cold and moisture the larvae become torpid. As pupae they exist about 11 to 16 days; hence the interval which elapses between the date of the depositing of the ovum and the maturity of the insect is ten or eleven weeks. The average life of flies reared from pupae was eight days. Some quarters situated within 20 yards of a bastion wall which was believed to harbour sandflies, were infested with them, but a barrack-block forty or fifty yards away was free. Repairing and facing the rampart for about sixty yards in the neighbourhood of barracks which have been notorious for nearly a century, on account of the incidence of sandfly fever in the troops occupying them, have been the means of reducing the number of sandflies with which they were previously infested, and of preventing the fever. Vegetation does not appear to have any connection with the breeding places of phlebotomi, though trees and shrubs near a house may afford shelter for the mature fly.

C. B.

NEWSTEAD (R.). *Notes on Phlebotomus, with Descriptions of New Species.* Part 1.—*Bull. Entom. Research.* 1912. Dec. Vol. 3. No. 4. pp. 361-367.

In consequence of the slender differences which can be found among the group of Phlebotomi, great care must be exercised

in preserving specimens. Pinning so injures them that identification and microscopical examination are rendered impossible. The insects should be placed on a web-like layer of teased cotton wool contained in a strong shallow pill-box, but they must not be covered with wool, for such an addition breaks the appendages. The author finds that the third segment of the palpi is provided with a compound group of minute modified spines, which are spathuliform with a long and strongly curved pedicel in *P. papatasi*; squamiform with a short pedicel in *P. minutus*; spathuliform with short pedicels in *P. squamipleuris*. Hirsute glands are present on the two segments before the terminal portion of the antennae of all species except *P. antennatus*. They appear as depressions fringed with fine hairs.

*P. minutus* exists throughout the Mediterranean area, and in India. Three specimens were obtained from Suda Island, Crete. (In the year 1909 every soldier of the British detachment stationed on this island was attacked with sandfly fever.)

The author thinks that an African variety of this species occurs in the Soudan, British Central and West Africa, and in Rhodesia, differing in the relatively longer second and fourth segments of the palpi, and in the more pointed wings, though he admits that considerable variations in these characters are observed.

A single specimen of a new species named *P. antennatus* was secured from Salaga on the Gold Coast in 1911: the bead-like form of the third to the thirteenth antennal segments separate it from other species, and it possesses shorter and stouter legs than *P. minutus*.

A new species from Khartoum, named *P. squamipleuris*, is distinguished by the pleurae being clothed with large flat mosquito-like scales.

Specimens of *P. papatasi* have been received from Cairo, and Tokar, Red Sea Province.

C. B.

NEWSTEAD (R.). *Phlebotomus* from West Africa.—*Bull. Soc. Path. Exot.* 1913. Feb. Vol. 6. No. 2. pp. 124-126.

A specimen was sent from Akjoucht, Mauritania which differed from *P. papatasi* in the inferior clasper being shorter and provided with four or more terminal spines, instead of two. It is probably identical with *P. duboscqui*.

A new species was obtained from Aguagon, Dahomey. The antennae indicate that it is a form intermediate between *P. papatasi* and *P. antennatus*. The third segment of the antenna is very short, and the end segment possesses hair-like scales.

C. B.

ROUBAUD (E.). Quelques Mots sur les Phlébotomes de l'Afrique Occidentale Française.—*Bull. Soc. Path. Exot.* 1913. Feb. Vol. 6. No. 2. pp. 126-128.

*P. duboscqui* is first seen at Akjoucht, Mauritania, in May at the beginning of the hot weather. It is also present in great

numbers at Boutilimit and Hombori. *P. papatasii* is abundant in the district of Timbuctoo, and is most numerous in newly built houses, in the clay walls of which it probably breeds. The natives are immune apparently, as no sandfly fever has been reported in that locality.

At Bingerville a lizard, *Agama colonorum*, asleep on a wall, was covered with *P. minutus*; the females were engorged with blood.

On the whole, phlebotomi do not appear to have a very extended range in West Africa, but it is possible they escape observation through feeding on animal hosts rather than on man.

C. B.

LUTZ (A.) & NEIVA (A.). *Contribuição para o Conhecimento das Especies do Genero Phlebotomus existentes no Brazil.*—*Zur Kenntnis der brasilianischen Phlebotomusarten.*—*Memorias do Instituto Oswaldo Cruz.* 1912. Vol. 4. No. 1. pp. 84-95.

Few specimens of phlebotomi have been captured in the towns of Rio, Bahia, and San Paulo. They are found in larger numbers in the woods of Rio Trombeta (Para), in the valley of the Maquine and on the banks of the Tiete.

They are known in the last district under the name "birigui." They are most abundant at heights 500-1,500 metres above sea-level, where they also occur.

The Brazilian phlebotomi are not identical with those from other parts of the world. The authors describe the following new species.—

*Phlebotomus squamiventris*, so named since the dorsal surface of the abdomen is covered with scales. The order of the segments of the palp beginning with the shortest is 4, 5, 3, 2. Its habitats are along the Rio Trombeta where it is known as "tatuquira"; and in Para.

*P. longipalpis* is characterised by the great length of the last segment of the palps, the index of which is 4, 2, 3, 5. It is found in the States of St. Paul and Minas.

*P. intermedius*, palp index 5, 4, 3, 2, occurs in the States of Rio de Janeiro, Minas, and St. Paul.

None of these species resemble *P. vexator* (Coquillett) of the United States, nor *P. rostrans* (Summers) from Rio Javary.

C. B.

SUMMERS (Sophia L. M.). *A Synopsis of the Genus Phlebotomus.*—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. No. 2. pp. 104-116. With 2 text-figures.

*Phlebotomus* is represented by some species in most parts of the tropical and sub-tropical world.

*P. papatasii* occurs in Europe, Asia, and Africa.

*P. mascitti* resembles *P. papatasii* except in the form of the spines of the upper clasper.

*P. minutus* is distinguished by the second segment of the palps being half the length of the third; by the position of the forks of the second longitudinal vein of the wing; and by the possession of four spines on the end segment of the upper clasper of the male. It is found in Europe, Asia, and Africa.

*P. nigerrimus*. NEWSTEAD separates this from *P. papatasii* by its colour, shape of wing and shorter 6th longitudinal vein.

*P. major*, described by ANNANDALE, and named by NEWSTEAD *P. perniciosus*, has shorter legs, and a smaller genital armature than *P. papatasii*. It is the common sandfly of Malta. It is also found in Asia.

*P. argentipes* is provided with black bristly hairs on its abdomen; its legs are very long; the distal joint of the upper appendages in the male is shorter than the proximal. It is an Indian species.

*P. himalayensis* is yellowish grey; the anterior branch of the second longitudinal vein is not much shorter than the second branch, and is four times as long as the distance between the forks. The superior appendages of the male bear three pointed chaetae.

*P. malabaricus* is provided with four chaetae on the upper male appendage.

*P. vexator* is a yellow species with brown mesonotum and hairs; the first submarginal cell is rather more than twice as long as its petiole; the arrangement of the five spines on the upper clasper of the male differs somewhat from that of *P. papatasii*. It is found in Plummerville Island, Maryland.

*P. cruciatus* is distinguished from *P. vexator* by its yellow hairs and first submarginal cell, which is three times as long as its petiole. It occurs in Guatemala.

*P. rostrans* is remarkable for the great length of its head. It has been captured on the Rio Javary, S. America.

*P. babu* was described by ANNANDALE who now identifies it with *P. minutus*.

*P. zeylanicus* closely resembles *P. argentipes*.

Other species are given in the preceding abstracts.

C. B.

## DENGUE.

STITT (E. R.). *Dengue, its History, Symptomatology, and Epidemiology*.—*Bull. Johns Hopkins Hosp.* 1913. April. Vol. 24. No. 266. pp. 117-121. [Discussion pp. 124-125.]

The author traces the history of dengue back to 1779, when GABERTE described an epidemic in Cairo. In 1780 a disease called "breakbone fever" was epidemic in Philadelphia; this was described very fully by RUSH who, though giving a clear picture of dengue, possibly included some cases of typhoid, yellow fever, malaria, and dysentery. In an outline of the symptoms of dengue, the author lays great stress on the early leucopenia, and the slowing of the pulse, even as low as 45 per minute; these conclusions were founded upon observations of 100 cases treated in the Philippines. It would appear however that this marked slowing of the pulse is less evident in America than in the Philippines, for GOLDBERGER stated in the discussion following the paper that it was not a marked feature in his experience, and though slower than in cases of influenza etc. the pulse was less slow than in cases of yellow fever. The mosquito experiments of ASHBURN and CRAIG are criticised. As strong evidence of the truth of the mosquito theory, the disappearance of dengue from Port Said in 1906-1907 during E. H. Ross's anti-mosquito campaign is brought forward. Also, when the author was in charge of a hospital in the Philippines, where 200 cases of dengue were treated in screened cubicles in a general ward, no infection of other cases in the ward took place.

P. W. Bassett-Smith.

CANAAN (T.). *Beobachtungen bei einer Denguefieber-Epidemie in Jerusalem.* [Observations on an Epidemic of Dengue in Jerusalem.]—*Arch. f. Schiff- & Trop.-Hyg.* 1913. Jan. Vol. 17. No. 1. pp. 20-25.

During the hot sirocco days of October 1912 this disease was very common in Jerusalem, spreading from the central parts of the city to the outskirts, affecting in the households almost all the members except children under two-and-a-half years. A full description of the clinical features is given; the most noticeable of these were, a short irregular fever, severe pains and great mental depression with or without delirium, marked gastro-intestinal symptoms, tendency to haemorrhages, and a scarlatiniform rash. The blood examinations always showed a pronounced leucopenia with a relative increase in the mononuclear cells; the parasites described by GRAHAM were never found. *Culex pipiens* are present in large numbers in Jerusalem.

The author states that this dengue epidemic in its symptoms resembled very closely pappataci fever (*Phlebotomus papatasi* is also very common in the town), but differed in the following points. Pappataci fever generally infects new comers, dengue the older inhabitants; in the former the fever lasts 3-5 days, in dengue 6-8 days, except in very slight cases. In the described epidemic the eruption was present in most of the cases, whereas in pappataci fever the eruption is present in only 30 per cent. of cases.

P. W. B.-S.

HOSSACK (W. L.). *The Problem of Dengue, Three-day and Seven-day Fever.*—*Indian Med. Gaz.* 1913. Feb. Vol. 48. No. 2. pp. 49-52.

The author gives a review of the work of ROGERS, MACCARRISON, FOOKS, ASHBURN and CRAIG, MEGAW, CHARLES, and KENNEDY relating to three day fever, seven day fever, and dengue in India and the Philippines. He describes the course of the epidemic in 1912 in the Calcutta district, with special reference to its occurrence on board ships, notably the "Lady Fraser." Details of a few cases which came under his own observation on shore are given in which the symptoms were severe and the pains intense, like those described in text books as associated with true dengue. He comes to the following conclusions:—

- (1) That the epidemic at Calcutta in 1912 was similar to that described as dengue in previous years.
- (2) That the 1912 epidemic comprised cases clinically distinguishable from three day fever but indistinguishable from seven day fever.
- (3) That the evidence is against phlebotomous being the infecting agent, and that in the later stages of the "Lady Fraser" epidemic, insect infection was almost precluded altogether.
- (4) That the recent epidemic at Calcutta seems to be an expression of a variable disease, caused by an ultra-microscopical organism which is endemically present in Calcutta, and gives rise to diseases known as three day fever and seven day fever.

P. W. B.-S.

SMITH (F.). **Dengue Fever among the Troops in Calcutta; its Identity with Seven-day Fever and Three-day Fever.**—*Jl. R. Army Med. Corps.* 1913. April. Vol. 20. No. 4. pp. 453-458. With 12 temperature charts.

The author lays great emphasis on the increase of mild local fevers in Calcutta from year to year. The common types found were previously called three day fever and seven day fever; these had all the clinical characters of the cases in the more extensive epidemic of 1912 observed by the author, who is sure that the disease is true dengue, and that therefore three day fever, seven day fever, and dengue, are here the same disease. The fever always comes on in the hot weather and ceases about October. Among the troops there were 113 cases in 1910, 124 in 1911, and 461 in 1912.

The author states that sandfly fever is common in the North West Provinces, and that troops when removed from that district to Calcutta had no immunity from the local dengue fever of Calcutta, which should have been observed if these fevers were due to the same cause; also that sandflies are not common in Calcutta, but very common in the North West Provinces. A series of cases and 12 charts are given, several of the latter giving a typical saddle-back type.

P. W. B.-S.

HARNETT (W. L.). **The Differential Blood Count in Dengue.**—*Indian Med. Gaz.* 1913. Feb. Vol. 48. No. 2. pp. 45-59. With 18 charts.

The author describes an epidemic of dengue fever which occurred in the lower Brahmaputra district from May to September 1912. Clinically the fever showed variations from the three day type to the saddle back and irregular continued type. Headache and pains in the loins were generally present, and rash was frequent, being most easily seen in Europeans. Specially attention was paid to the changes in the blood; besides the usual leucopenia, diminution in the polynuclear leucocytes, and a relative increase in the mononuclear lymphocytes, a marked eosinophilia was recorded, giving an average of 13·8 per cent. in the 24 cases; the details of each are given. The estimation was made from a count of 200 leucocytes in most cases. Most of the patients were Asiatics, but the author states that in several examinations for intestinal ova negative results were obtained, and these same characters were found in a number of blood examinations which were made on patients suffering from "three day" fever at Dehra Dun. The author considers that leucopenia with a rapidly developing eosinophilia is a characteristic blood change found towards the end of the first week of an attack of dengue, and that this peculiarity may help to settle the question whether "three-day fever" "seven-day fever" and dengue are one and the same disease. Further observations are however required.

[The number of leucocytes counted is small to dogmatise on, and other causes of eosinophilia are hardly sufficiently excluded.]

P. W. B.-S.

## SLEEPING SICKNESS.

## TRANSMISSION.

KLEINE (F. K.) & ECKARD (B.). Ueber die Bedeutung der Speicheldrüseninfektion bei der Schlafkrankheitsfliege (*Glossina palpalis*). [The Significance of Infection of the Salivary Glands in Sleeping Sickness Tsetse Flies.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. Apr. Vol. 74. No. 1. pp. 183-186.

The object of the experiments described in this paper was to ascertain whether the trypanosomes found in the salivary glands and in the alimentary canal of *G. palpalis*, proved to be capable of infecting animals with *T. gambiense*, were virulent when injected into susceptible animals. If only those animals into which the gut contents were injected became infected, then it would appear that the salivary gland invasion was only an incidental occurrence and not essential. This would also be the case if both sets of animals became infected. If however infection only occurred in those animals which had received the salivary gland contents, some special significance must be attributed to these organs; the completion of the development of the trypanosome must take place only in the salivary glands.

*Experiments.*—Six infective *Glossina palpalis*, 45-62 days old, were killed and the contents of the salivary glands and intestine injected separately into different monkeys; in the case of three of these the contents of the proventriculus were also injected into healthy monkeys. Only those animals became infected which had received the contents of the salivary glands; those which had been injected with the trypanosomes from the gut or proventriculus remained unaffected.

It follows that infective parasites are only found in the salivary glands and that before development is complete the trypanosomes invade these structures. So long as it was believed that salivary gland infection occurred only occasionally it was assumed that the trypanosomes reached the glands via the proventriculus, oesophagus and hypopharynx; since however it appears to be the rule, this route of invasion seems unlikely and we must consider the direct path through the coelom. Experiments to determine the point are in progress. [These results are at variance with those of the Royal Society Commission in Uganda, and of KINGHORN and YORKE in Northern Rhodesia; both these sets of observers found that the intestinal parasites of infective flies were virulent as well as the salivary gland contents.]

W. Yorke.

WENYON (C. M.). Experiments on the Transmission of *Trypanosoma lewisi* by means of Fleas.—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. No. 2. pp. 119-123.

The author refers to a previous paper (this *Bulletin*, No. 7, p. 366) in which he reported he had been able to confirm NÖLLER's work (*Sleeping Sickness Bulletin*, Vol. 4, p. 215) on the transmission of *T. lewisi* by means of the dog flea *Ctenocephalides canis*. An infected flea, fixed on wire as recommended

by NÖLLER, was fed upon a clean rat and the faeces passed during the feed were caught on a coverslip and transferred by means of a fine pipette to a tube of N.N.N. medium. The diluted fluid was then introduced into the mouth of another clean rat, care being taken to avoid injuring the skin. The experiment was repeated three times with the dog flea and in each instance it was the rat which received the faeces that became infected and not the rat on which the flea had fed.

This experiment was repeated with the human flea, *Pulex irritans*. An attempt was also made to transmit *T. lewisi* by means of *Pulex irritans* when free. On three occasions fleas, proved to be infected by examination of the faeces, were liberated from the wire and placed on clean rats. In each instance the flea disappeared and the rat did not become infected. It was subsequently found that *Pulex irritans* when placed on a rat exhibits great uneasiness, jumping on and off the animal, giving the impression that it does not feel at home. Its behaviour was in marked contrast to that of the true rat fleas *Ceratophyllus fasciatus* and *Xenopsylla cheopis*, which immediately hide themselves in the fur.

Experiments conducted with *Xenopsylla cheopis*, the common rat-flea of India, demonstrate that this flea is a true host of *T. lewisi*. The results obtained with *Xenopsylla* indicate that probably the faecal mode of infection obtains as in the case of the dog and human fleas.

The author refers to the experiments of MINCHIN and THOMSON with *T. lewisi* and *Ceratophyllus fasciatus*, which suggest that infection results from the regurgitation of the small trypanosomes which have wandered forward from the rectum to the mid-gut; he points out that this cannot be the regular mode of infection in *Ctenocephalus* and *Pulex*. The passage forwards of the small trypanosomes from the flea's rectum to the mid-gut is in the author's opinion fortuitous, dependent upon the violent peristaltic contractions of the gut of the flea whilst feeding. It is urged that the fact that the final development of the infective forms of *T. lewisi* occurs in the rectum strongly supports the faecal method of infection. In *Glossina* and leeches, which are known to transmit trypanosomes by their bite, the final development of the parasites is in the anterior part of the gut or in its appendages (proboscis or salivary glands).

The favourite haunt of *Xenopsylla* is the posterior part of the rat's back, just above the root of the tail. Here the faeces are deposited and the rat licks up the dry faeces and occasionally the moist faeces just deposited. When a flea is quite undisturbed while feeding it will often become distended to an enormous extent; in this condition a slight movement will cause it to eject at once a comparatively large drop of faeces and the rat has thus every chance of taking up freshly passed moist faeces. It is suggested that, as *T. lewisi* can complete its development in so many different invertebrate hosts, other trypanosomes may follow in this respect. In conclusion the author remarks that it cannot any longer be held that a difference in invertebrate host necessarily means a difference in species of trypanosomes.

W. Y.

C



## CLINICAL.

LAGANE (L.). Pouls lent dans la Trypanosomiase humaine.—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 269-272.

An account is given of two cases of human trypanosomiasis from the Congo in which bradycardia was a pronounced symptom. The pulse frequency was in the first case 50-58 per minute and in the second 50-62. With the exception of bradycardia auscultation of the heart revealed nothing abnormal. Administration of atropine or nitrite of amyl accelerated the pulse. The inference is drawn that the condition is not due to auriculo-ventricular inco-ordination, but that it is a true nervous bradycardia. It is suggested that possibly the phenomenon may result from slight irritation of the vagus, as it passes through the sub-arachnoid space. The symptom is unfavourable from the point of view of prognosis, whilst its disappearance under the influence of treatment is to be regarded as a sign of amelioration.

W. Y.

HECKENROTH (F.). Réactions locales de début dans un nouveau Cas de Trypanosomiase humaine chez l'Européen.—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 267-269.

Reference is made to a case of trypanosomiasis in which the first symptom was a painful swelling on the scalp. The following day a similar swelling was observed on the inner surface of the left knee. The author states that he has no doubt that these resulted from the bites of an insect, most probably the tsetse as this fly is abundant at M'Pokou (Congo) where the patient was stationed. The diagnosis of the disease was made subsequently by examination of the juice from one of the enlarged cervical glands. He concludes by stating that such local reactions should make Europeans suspect trypanosomiasis and induce them to present themselves for medical examination with as little delay as possible. [Such furuncle-like swellings have frequently been attributed to infective bites. This case is of interest in so far as there were simultaneously two lesions situated on such widely separated portions of the body as the scalp and the knee.]\*

W. Y.

NEWHAM (H. B.). Trypanosomiasis. London School of Tropical Medicine. 3rd Report.—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. No. 2. pp. 144-146.

This paper gives the clinical and pathological record of a case (No. 16, W.G.) of trypanosomiasis contracted in Rhodesia. (See *Sleeping Sickness Bulletin*. Vol. 4. p. 19.) During the last six weeks of life cerebral symptoms were pronounced.

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\* Those interested in this symptom should refer to *Sleeping Sickness Bulletin* Vol. 3, pp. 5-7, where the cases then recorded as showing it were collected and discussed. One patient had no less than five, two others two of these swellings. The symptom seems to be less usual in natives than in Europeans. A.G.B.

Microscopical examination of the cerebrum revealed well-marked perivascular infiltration with round cells; a similar but less extensive infiltration was noted in the grey matter of the spinal cord. The author remarks that this is the third case of Rhodesian trypanosomiasis which has been under observation at the London School of Tropical Medicine. The other two died five months after infection, without the development of cerebral symptoms. This patient lived three years and three months. In spite of the fact that treatment had been commenced within a month of the onset of symptoms the disease had never been more than under control. The contrast between these three cases and those from other parts of Africa indicates the greater malignancy of the Rhodesian infection.

W. Y.

## TREATMENT.

AUBERT (P.) & HECKENROTH (F.). *L'Arsenophénylglycine dans la Prophylaxie chimique de la Trypanosomiase humaine.*—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 272-276.

Reference is made to a previous paper (*Sleeping Sickness Bulletin*, Vol. 3, p. 259) in which, on account of the intense local inflammation which frequently followed subcutaneous injection of arsenophénylglycin, the authors recommended intravenous injection of the drug. This paper records the results of treatment of a large number (88) of cases of sleeping sickness by intravenous injection of this substance. Certain recommendations regarding the general technique are made. Owing to the rapidity with which arsenophénylglycin decomposes in air one should always make certain that there is a vacuum in the ampoules in which the drug is supplied. The drug was dissolved in water containing 6 to 9 per cent. of sea salt or merely in distilled water. The freshly made filtered solution should be used. One or two injections were given. The dose recommended is 45 milligrammes per kilo of the body weight of the patient. The two injections should be given at intervals of four or five days. Larger doses should be avoided, as in certain cases they may produce toxic effects. The degree of concentration of the solution is important. The volume of fluid injected should not be less than 100-120 cc. If more concentrated solutions are used reactions are more common.

In a table the results are given of treating by this method 37 cases in which it had been possible to make frequent examination of the centrifuged blood subsequently. Twenty were improved by the treatment, twelve remained unaffected, and in five the symptoms were aggravated. Four of the patients died. The duration of sterilisation in 25 was at least 6 months, and five were in a state of perfect health 25, 24, 22, 18 and 15 months respectively after treatment and can, therefore, be considered to be definitely cured.

The conclusions are:—

Arsenophénylglycin is a remedy which possesses an energetic and prolonged power of sterilisation.

Intravenous injection is the best method of administering the drug.

Injections given in the doses mentioned are tolerated well by the patients.

On account of the promptness of its action, the prolonged sterilisation, and marked amelioration of the general condition which it causes, intravenous injection of arsenophenylglycin is a valuable method of treatment and one which it is to be hoped will be generally used in the treatment of human trypanosomiasis.

W. Y.

**YOUNG (J. C.). A Case of Trypanosomiasis treated by Intravenous Injections of Tartar Emetic.**—*Univ. of Durham College of Med. Gaz.* 1912. Nov. 15. Vol. 13. No. 2. pp. 19-25.

The author, as ship's surgeon, had charge of a European suffering from trypanosomiasis between Chinde and Marseilles. This was the elephant hunter whose case was originally described in *Sleeping Sickness Bulletin*, Vol. 4, p. 266. Further accounts of the case have been given in this *Bulletin*, Vol. 1, pp. 275 and 514. On the patient's embarkation the chief points noted were the extreme pallor of the face, emaciation, and oedema of feet and legs. Treatment was by intravenous injections of tartar emetic which appeared to give rise on several occasions to oedema of the middle third of the upper lip and to extreme distention of the abdomen. Six injections were given at intervals of five days and were followed by marked improvement. The temperature chart is given. When the ship was in the Red Sea it was noticed that the axillary and femoral glands became enlarged and slightly tender.

A. G. B.

#### TRYPANOSOMIASIS IN RHODESIA.

**STOHR (F. O.). Report on Sebungwe Fly Area.** MS. Report to British South Africa Company. Dated Jan. 1913.

After giving an account of the physical characters of the Sebungwe fly belt (Southern Rhodesia), the author states that he has found it impossible to correlate the boundary of the fly area with any physical feature. Game is numerous and as a general rule the fly is thickest where the game is most abundant, but at Bwampu where the fly is very plentiful there seems to be no game in the dry season.

The area, which is roughly 2,500 sq. miles, is sparsely populated, there being only about 3,000 natives; with the exception of a few Government officials there are no white people.

*History.*—It is generally agreed among the old chiefs that before the rinderpest fly was more numerous than at the present time, and all informants connected its disappearance with the extermination of the buffalo. Soon after the reappearance of the tsetse domestic stock began to die.

In all 2,340 persons were examined and 11 cases of Sleeping Sickness were found. The diagnosis in nine of these was made by gland puncture (the glands of 87 persons were punctured) and in the other two by examination of the blood (170 blood examinations were made); the first of these two cases was an infant with little shotty glands and the second was one of the author's carriers in whom trypanosomes were found on the first day of sickness

The clinical features of the disease are compared with those of cases seen by the author in Katanga. It is noted that this form is of much greater severity than that of the Katanga. Of the 11 cases found one had a history of 2½ years' illness, whilst that of the others was less than 6 months. The inference is drawn that the disease rarely lasts more than a year. Nine of the patients were obviously ill when the diagnosis was made. Five had the distinctive chain of glands, whilst other five had only one or two enlarged glands. In every case trypanosomes were easily found at the first puncture.

*Trypanosomiasis of domestic animals.*—Trypanosomes were found in 2 of 51 goats, 6 of 24 sheep, and in 12 of 20 dogs. Only animals in obviously poor condition were examined.

*Trypanosomiasis in game.*—Blood was examined from the following wild animals shot in the fly area—duiker (1), elephant (1), roan (1), zebra (4), eland (4), mpala (2), warthog (2), and waterbuck (5). Trypanosomes were found in two waterbuck. [A description of the trypanosomes is given—the author assumes that the parasites infecting man, small stock and dogs are the same—but as the examination was limited to fresh preparations no conclusion can be drawn as to the identity of the trypanosome or trypanosomes found. An account of the parasites obtained from one of the human cases, from an infected dog and goat, and from waterbuck is given below by BEVAN.]

Discussing the question of prophylaxis the author writes that he considers that if the population be removed from the area, the fly and game are likely to increase and spread until the invasion of surrounding villages demands further removals. He is of the opinion that some measure should be undertaken to deal with the fly or the game, which was shown by KINGHORN and YORKE in N. Rhodesia to be the reservoir of the virus. As we have no knowledge how satisfactorily to attack the fly, he advocates the shooting of the wild animals in the fly area.

W. Y.

BEVAN (Lt. E. W.). Preliminary Notes on a Trypanosome causing Disease in Man and Animals in the Sebungwe District of Southern Rhodesia.—*Jl. Trop. Med. & Hyg.* 1913. Apr. 15. Vol. 16. No. 8. pp. 113-117. With temperature charts and 2 plates.

The strains used in these observations were obtained from natives and animals suffering from trypanosomiasis in the vicinity of the Busi river in the Sebungwe District of Southern Rhodesia (see above). The blood from the natural cases was inoculated into clean rabbits which were returned without delay to the Veterinary Laboratory at Salisbury. Two rabbits were inoculated from man, two from a dog, and two from a goat. A table of observations made on these six rabbits is given in some detail. Four of them died in from twelve to twenty-seven days, and two, infected from the dog and goat respectively, had a chronic infection. Another table gives the duration of the disease in animals subinoculated from the rabbits. The movements of

the trypanosomes of the three strains were similar; there was no active translation. The fixed and stained trypanosomes of each strain appeared identical. There were tadpole forms of 'pecorum' type, stumpy, intermediate, and long free-flagellated forms of 'nagana' type, the types merging into one another. A number of individuals of each of the strains showed numerous large metachromatic granules, both in the anterior and posterior halves of the body in all forms of the parasite. In each strain forms were met with having the nucleus situated in the posterior third. Measurements of the trypanosomes of each strain are given. It is seen that the Sebungwe trypanosome in the rabbit shows shorter minimum and maximum forms than allied species, but that the average corresponds very closely with KINGHORN and YORKE's estimate of *T. rhodesiense*.

The symptoms seen in rabbits, dogs and sheep are given. The rabbits did not show oedema of the head characteristic of the infection produced by the W. A. strain of Luangwa trypanosome. There was some oedema of the face in sheep, but it did not increase to the same extent as did that of sheep inoculated with the Luangwa trypanosome. The temperature charts of these animals are given. Blood smears taken from wild animals shot in the Sebungwe fly area were stained and examined at the laboratory. Trypanosomes were met with in smears from three waterbuck. No distinction could be drawn between these trypanosomes and those met with in the infected natives and domestic animals.

The plates show trypanosomes seen in a blood smear taken from a naturally infected dog, and sheep infected one with the original 'W.A.' strain and the other with the Sebungwe strain of *T. rhodesiense*.

A. G. B.

WENYON (C. M.) & HANSCHALL (H. M.). **A Further Note on *Trypanosoma rhodesiense* from Three Cases of Human Trypanosomiasis.\***—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. No. 2. pp. 123-128.

This paper records observations on the incidence of posterior nuclear forms in the blood of man and rats infected with *T. rhodesiense*. No such forms were found in the blood of any of the three human beings (G, E, T) examined, although in one of the cases the trypanosomes occurred in the blood in rather large numbers. They were found, however, in the blood of the first rat inoculated from the human cases. Observations were made with the object of determining in the subinoculated rats the constancy of the appearance of the posterior nuclear forms, their percentage number and the extent of variation of this number, and their relation to the virulence of the strain. Although the nuclear displacement occurred most frequently in the broad trypanosomes, it was also observed in the narrowest forms with long free flagellum.

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\* For a summary of the previous note see this *Bulletin*, Vol. 1, p. 276.

On certain days of the infection in rats posterior-nuclear forms were absent: this applies to each of the three strains. In the fifth passage of one of the strains (E), during the twelve days the rat lived no posterior nuclear forms were found. In all the strains, and in some particular passages, the percentage of these forms varied greatly. The authors have the impression that posterior nuclear forms are more numerous in the first passage in the rat, and that they tend to become scantier with each subsequent passage.

No correlation was found to exist between the number of posterior nuclear forms and the virulence of the strain for white rats.

Details of the percentage of posterior nuclear forms encountered on various days of the infection in the different passages in white rats in each of the strains are given in tables.

W. Y.

VON PROWAZEK (S.). Ueber reine Trypanosomenstämme. [On Pure Strains of Trypanosomes.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Apr. 16. Vol. 68. No. 5-6. pp. 498-501.

The author refers to the work of OEHLER (see this *Bulletin*, Vol. 1, p. 525). Similar experiments have been carried out at the Institut für Schiffs- und Tropenkrankheiten at Hamburg. The strains used were one of *T. rhodesiense*, one of *T. equinum* which was naturally atoxyl-fast,\* and the Proteosoma of the canary. The object was to see (1) Whether infection could be obtained with a single trypanosome; (2) Whether dimorphism, which is specially marked in *T. rhodesiense*, is primary, or whether the various forms can arise from a single individual; (3) To ascertain whether various peculiarities of a strain, such as atoxyl-fastness and absence of blepharoplast, are contained primarily in the strain or appear owing to selection or are mutation products; (4) To obtain by pure infection material for chemotherapeutic experiments; (5) To make curves of pure and non-pure strains after BRUCE's method; (6) To answer for bird malaria the question whether one can infect with ripe macrogametes and thus indirectly to show that relapses are referable to parthenogenesis. It is noted that these last experiments were negative.

(1) The author succeeded, as OEHLER did, in obtaining infection with a single trypanosome. (2) In the *rhodesiense* strain there appeared in the third, and certainly in the fourth passage broad forms as well as slim forms. Forms without nuclei were also seen at the fourth passage. The dimorphism of this trypanosome is accordingly not primary. (3) The experiments showed that the pure strain of mal de caderas behaved differently towards atoxyl and salvarsan to the original strain; in the former the trypanosomes disappeared for a time and death was delayed. (4) At the first divisions of the strain obtained from a single individual variations as regards the division products are seen. The trypanosomes do not take on vital staining

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\* For an account of this strain see this *Bulletin*, Vol. 1, p. 56.

simultaneously or equally quickly, nor do they all die at the same time. Differences are also observed in the method of division. Thus, early in the life of the pure strain, primary differences in metabolism, as well as differences in division, were obtained.

The naturally atoxyl-fast strain of mal de caderas at the time of these experiments had gone through 714 passages without passing through the body of an insect and had undergone no change. The experiments are still in progress.

A. G. B.

DOBELL (Clifford). **Some Recent Work on Mutation in Micro-organisms.**—*Jl. of Genetics*. 1912. Nov. 21. Vol. 2. No. 3. pp. 201-220.

The author uses the term mutation to denote those heritable modifications which have been induced in various ways in various micro-organisms. He begins with observations on mutations in trypanosomes, which, he writes, are considered in a somewhat critical spirit. They are classed as morphological and physiological. Under the heading Morphological Mutations the work of WENDELSTADT and FELLMER is first considered (see *Sleeping Sickness Bulletin*, Vol. 1, p. 388 and Vol. 2, p. 174). These authors found that *T. brucei* and *T. lewisi* may be inoculated into cold-blooded vertebrates and invertebrates, in which they live for a certain time and undergo morphological changes.\* He goes on to consider the morphological mutation discovered by WERBITSKI and confirmed by other workers (see *Sleeping Sickness Bulletin*, Vol. 2, p. 98, Vol. 3, pp. 221, 313, 314, 458, and Vol. 4, p. 33).

Under the heading Physiological Mutations the phenomenon of chemo-resistance is considered and some space is given to the work of GONDER on the loss of arsenic resistance of *T. lewisi* in the course of development in the louse (see *Sleeping Sickness Bulletin*, Vol. 4, p. 13). The more important results are summarised thus—

“(A) It has been stated that the passage of certain Trypanosomes, which normally occur in mammals, through cold-blooded vertebrates and certain invertebrates, causes them to undergo certain structural changes which persist during subsequent divisions (Wendelstadt and Fellmer). This work has not yet been confirmed.

“It has further been stated (Werbitzki) and confirmed (Laveran and Roudsky, Kudicke) that certain dyes can destroy a definite organ (kinetonucleus) in a Trypanosome, without killing or injuring it or impairing its power of propagation. Thus new races of Trypanosomes may be produced which completely lack this organ. It has, moreover, been rendered highly probable that the dyes which have this power possess a certain chemical structure (orthoquinoid substances of Ehrlich): and that the dyes have a specific action upon the kinetonucleus—but upon no other organ in the Trypanosome—and bring about its destruction by autoxidation (Laveran and Roudsky). New races of Trypanosomes are thus produced by modifying the individuals of the old—not by selection.

“(B) Races of Trypanosomes without kinetonuclei possess a lowered virulence (Werbitzki, Laveran and Roudsky).

“By the action of various drugs and antibodies, races of Trypanosomes may be obtained which are resistant to these substances (Ehrlich, Meñil

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\*In the second of the above references these trypanosomes are said to have been inoculated into adders; this should have been grass snakes (*Tropidonotus natrix*), as Mr. Dobell has pointed out.

and Brimont, Breinl and Nierenstein, Levaditi and Twort, &c.). These races subsequently breed true—though it may be a necessary condition of this that they be kept in the same sort of host as that in which they originally acquired their resistance.

"Races of Trypanosomes with a changed virulence are said to be produced by passage through certain animals (Wendelstadt and Fellmer): but this has been denied (Gonder and Sieber, Laveran and Pettit).

"By treating *T. lewisi* with arsenophenyglycin, a race may be obtained which is resistant to this drug. This race breeds true—retaining its resistance during numerous passages through untreated rats. Resistant and non-resistant races remain unchanged, as regards this character, when grown in artificial cultures. When the resistant race undergoes a development in the louse—the exact nature of which is not determined, though it is possibly sexual—resistance is gradually lost, and the race returns to the original non-resistant condition (Gonder).

"It has not been definitely determined whether resistance is brought about by the direct action of the poison on the living Trypanosome (Ehrlich, &c.), or whether it is the result of selection (Levaditi, &c.)."

A. G. B.

OGAWA (M.). *Quelques Observations sur le Dimorphisme de Trypanosoma pecaui*.—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Mar. 15. Vol. 68. No. 3-4. pp. 332-334. With 3 tables and 3 text-figures.

The author has made a short study of *Trypanosoma pecaui*, the virus of Baleri, on biometric lines, having measured 1,200 trypanosomes from two guinea-pigs. 1,000 trypanosomes at the rate of 100 a day for 10 days were taken from one guinea-pig, the remainder from another. Two forms of parasites occur in *T. pecaui*: (1) long drawn out forms, (2) short stumpy forms. The author states that during the course of infection a relation exists between the two forms. Long forms and dividing forms appear first in the blood, short forms not till some days after the long ones. When the disease is of somewhat long duration as in guinea-pigs, from the middle period short forms occur in large numbers, but long forms reappear and dominate during the last period. In animals which succumb quickly, the long forms are in much greater numbers than any other forms, throughout the infection.

Two tables, showing the percentage of trypanosomes of lengths varying by half a micron obtained by the use of the curvimeter of 1 mm. tooth interval ( $= \frac{1}{2}\mu$  at a magnification of 2,000 diameters), are given, together with a graphical representation.

The dimensions of the trypanosomes are as follows: Long drawn out forms,  $24\mu$  to  $34\mu$  by  $1\mu$  to  $1.5\mu$ . Short stumpy forms without free flagella  $12\mu$  to  $20\mu$  by  $2.5\mu$  to  $4\mu$ . Intermediate forms with short flagella  $21\mu$  to  $23\mu$  by  $1.5\mu$  to  $2\mu$ . A few forms with posterior nuclei were seen, of which 3 text figures are given.

H. B. Fantham.

CAZALBOU (L.). *Observation d'un Nouveau Trypanosome chez le Lapin*.—*Receuil de Médecine Vétérinaire*. 1913. Mar. 15. Vol. 90. No. 5. pp. 155-158.

The author has recently studied a trypanosome occurring in the blood of young rabbits at Rennes. The parasite was obtained



from a victim of an epizootic. When three weeks old, the growth of the rabbits was interrupted. They rapidly became emaciated, paralysis of the hind-quarters occurred and death ensued some weeks after the appearance of the malady. Investigation of the glands was negative. From the blood of one dead rabbit, fifteen slides were prepared and examination of these revealed the presence of one solitary trypanosome. Sub-inoculation of a guinea-pig with blood from a rabbit dying from the epizootic has so far been negative. The proof of the pathogenicity of the trypanosome has not been established.

The trypanosome is about  $80\mu$  long, the free flagellum being  $10\mu$  to  $12\mu$ . The undulating membrane is broad and the maximum width to the flagellar border is  $8\mu$ . The oval nucleus is median. One text-figure is given.

The author gives a succinct review of former records of trypanosomes found in rabbits, all of which differ from the organism described by himself by being much shorter. It also shows dissimilarities from the large trypanosomes of cattle such as *T. theileri* and *T. ingens* by dimensions, folding of the membrane and position of the blepharoplast.

Cazalbou calls the new parasite *Trypanosoma gigas*.

The origin of the parasite is quite unknown. There were no fleas nor lice on the healthy parents of the victims, and mosquitoes from the neighbourhood have shown no evolutionary forms of flagellates.

H. B. F.

**DOFLEIN (F.). Ueber Dauerformen und Immunität beim Frosch-trypanosom.** [On the Resistant Forms and Immunity of the Trypanosome of the Frog.]—*Berichte der Naturforschenden Gesellschaft zu Freiburg i. Br.* Bericht über die Sitzung am 19. Feb. 1913.—1913. Mar. Vol. 20.

The author reviews briefly his work on *Trypanosoma rotatorium* of the frog, together with that of OGAWA and MENDELEEFF-GOLDBERG. The main results of the research were as follows:—In the blood and internal organs of frogs, trypanosomes intermediate between the flagellate and nonflagellate stages were found. Cultures gave similar results. The nonflagellate forms appear to have lost the power of division. Very small non-flagellate forms about  $1\frac{1}{2}\mu$  in diameter occur in old cultures; they are resistant forms, resembling the latent forms of pathogenic trypanosomes, and are capable of starting new cultures. *T. rotatorium* in cultures shows physiological variation in regard to fat storage and osmotic relations, together with morphological variation. Much fat occurs as reserve food material in the small, cultural trypanosomes, a little fat only in the trypanosomes of frog's blood. The variations of blood and cultural forms are ascribed to adaptive processes. Transplantation of trypanosomes from blood to culture causes reappearance of the multiplicative capacity.

No disease symptoms occur in infected frogs, which usually contain few trypanosomes. By analogy with Texas fever, a

trypanosome infection may manifest itself heavily in the tadpole stage and become latent at metamorphosis, the adult thenceforth acting as a reservoir of the parasite.

Whilst the trypanosomes die but remain undissolved in the blood of toads, tree frogs, trout, leeches, guinea-pigs, rabbits, goats, and men, they are rapidly killed and dissolved in blood from infected frogs. The frog, then, is specifically immune to *T. rotatorium*. A substance is formed in the blood of infected frogs which reacts on cultural trypanosomes but not on the large blood trypanosomes. Intraperitoneal inoculation of tadpoles is easy. The tadpole can be observed for 14 days. In them the trypanosomes enlarge and gradually assume a form similar to the large blood trypanosomes.

The power of immunity of the trypanosomes in the blood may be associated with the firm structure of the pellicle and surface protoplasm of the organism. The membrane formation is thought to be dependent on the amount of fat in the body, and its variation in form in blood and cultural trypanosomes is due to the diversity in maximum growth and capacity for division. The acquisition of immunity is said to be connected in some way with the appearance and growth of the membrane.

H. B. F.

VON SCHUCKMANN (W.) & WERNICKE (K.). **Einiges über Methoden und Ergebnisse der Trypanosomenzüchtung.** [Methods and Results of Trypanosome Cultivation].—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Mar. 1. Vol. 68. No. 2. pp. 241-255. With 1 text-figure.

The authors sought to improve on the medium and method of MACNEAL and NOVY because of the difficulty of obtaining rabbits, rats, guinea-pigs and other experimental animals, and the danger of contamination of the medium, especially during defibrination.

They made a large number of experiments. When the medium was ready the tubes were closed with melted paraffin, in order to prevent evaporation of the water of condensation, an improvement on the india-rubber caps used by NOVY. They consider that the size of the tubes generally used is disadvantageous, the large quantity of air favouring evaporation; they therefore employed serum diagnosis tubes 10 cm. in length and 13 mm. in diameter. Systematic experiments were made with various kinds of blood and with mixtures, five parts of serum of one animal being mixed with one part of blood corpuscles from another. Rabbits, guinea-pigs, goats, and cattle were used. It was seen in the sequel that no kind of serum or blood was especially favourable to the growth of the trypanosomes used, almost exclusively those of birds, especially the wood owl and the little owl; *T. lewisi* was also employed. With regard to cattle, trypanosomes have been several times cultivated from their blood; it is therefore necessary after the tubes have been prepared to sterilise them by fractional sterilisation. The authors have not used the method of MATHIS, who heated his tubes to 120° C.

Sixty experiments are set forth in a table, in which is given the composition of the medium, the age of the medium, the number of tubes inoculated, the result, the duration of life of the culture, and the number of generations for which it was continued. The organisms used were partly the so-called *Halteridium* culture, and later *T. avium* from the little owl.

Lastly they made experiments to test the correctness of SCHAUDINN's view. They did not succeed in obtaining any proof of a genetic connection between *Haemoproteus* and *Leucocytozoon* on the one hand, and the so-called *T. avium* on the other. They doubt the correctness of the conclusion which M. MAYER formed from his researches (*Archiv f. Protistenkunde*. 1911. p. 232).

A. G. B.

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## PROTOZOOLOGY.

DOFLEIN (Franz) & KOEHLER (Otto). *Ueberblick über den Stamm der Protozoen*. [Survey of the Protozoa.]—*Handbuch der pathogenen Mikroorganismen*, Kolle u. Wassermann. 1912. 166 pp. 99 text-figs. (Verlag von Gustav Fischer in Jena.)

The article is only intended to be a short review of the Protozoa as a whole, and occupies 147 pages of text, followed by references to literature more or less complete. There is first a general account of the biology and morphology of the group, extending over 25 pages. The Protozoa are divided into (1) *Plasmodroma*, including Mastigophora, Sarcodina and Sporozoa and (2) *Ciliophora*, including Ciliata and Suctorina. Six and a half pages are devoted to the Trypanosomidae, including *Trypanosoma*, *Herpetomonas*, *Schizotrypanum*, *Endotrypanum* and *Leishmania*. The life-cycle of *Entamoeba coli* is illustrated by HARTMANN's well known diagram. A list of other species of Entamoebae is given.

The Sporozoa are well treated and illustrated in the sixty pages devoted to them. The last few pages contain a short account of the Ciliophora.

[While the article may appear disappointing to the student of tropical medicine, it must be remembered that other articles in the Handbuch are specially devoted to pathogenic Protozoa.

We must protest against the "Germanising" of the spelling of group and generic names. It is inaccurate to replace *c* by *k* in such names as Sarcosporidia, Macrostoma, Leucocytoagarina, Microsporidia, Actinomyxidina and Actinosphaerium.]

H. B. Fantham.

RONDONI (Pietro). *Sulla Classificazione dei Protozoi Emoparassiti: il Nuovo Ordine dei Binucleati* (Hartmann). [Classification of Protozoal Blood Parasites. Hartmann's Binucleata.]—*Lo Sperimentale*. 1913. Apr. 7. Vol. 67. No. 1. pp. 105-118.

This paper is a review of the Binucleata, a group instituted by HARTMANN for Flagellates possessing a nucleus and a blepharoplast. The author discusses the genera *Trypanosoma*, *Trypanoplasma*, *Herpetomonas*, *Crithidia* and allied forms, and their possible phylogeny. He mentions the blepharoplastless trypanosomes of WERBITZKI. He also discusses the relationship of the Spirochaetes to the Binucleata. He states that the group, though useful, is an artificial one, a fact which is now generally recognised.

H. B. F.

i. FRANCHINI (G.). *Un Nouveau Protozoaire parasite de l'Homme provenant du Brésil*.—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 156-158.

ii. FRANCHINI (G.) & MANTOVANI (Mario). *Di un Nuovo Parassita Protozoario trovato nel Sangue Periferico ed Epatico in un Caso di Infezione cronica mortale proveniente dal Brasile*. [New

Protozoal Parasite in the Blood of Man.]—*Ann. di Med. Navale e Coloniale*. 1913. Feb. Anno 19. Vol. 1. No. 2. pp. 125-138. With 1 coloured plate.

i. A new parasite has been found by Franchini in the blood of an Italian physician, who had resided in Brazil for 18 years. The man had enjoyed good health till three years ago when he noticed a general feebleness set in, but without loss of appetite or loss of weight. A year later, a tumefaction, hard and indolent, becoming as large as a pigeon's egg, formed in the right lateral cervical region of the neck. It was removed but the wound did not close, and a white non-purulent secretion appeared. Soon after, another tumefaction appeared in the same place. He returned to Italy, became anaemic, suffered from fever and entered hospital.

The fever, at first irregular, became almost quotidian, was preceded by shivering and followed by great perspiration. The spleen was somewhat enlarged, the liver gradually extended to the umbilicus, and in the right lobe a cyst of the size of an orange, which fluctuated, formed. Puncture of the cyst gave a thin, reddish liquid. On examination of the patient great anaemia and emaciation were observed; the tumefaction in the neck was the size of a nut and in contiguity with the lymphatic glands; lymphatic glands normal; urine with traces of albumin; blood showing acute anaemia, with leucopenia, no eosinophilia; negative Wasserman reaction; nothing special noted on spinal puncture. Blood examination gave neither malarial parasites, leishmania, spores, nor fungi. Cultures of blood on media appropriate to Blastomycetes were negative. In numerous smears of blood taken by finger puncture, a parasite, often free in the plasma, sometimes within erythrocytes and very rarely in leucocytes, occurred. It was found also in material from three liver punctures. It was present in the peripheral blood only during the febrile attacks. The parasites were hardly visible in fresh blood and did not seem to move. Smears stained with Giemsa showed oblong or pyriform parasites with black pigment, often with two masses of chromatin, one large and one small, and protoplasm containing vacuoles and chromatic granules. Parasites of the size of a red corpuscle, rounded, with several chromatic masses and one or two centrosomes were probably forms showing multiple division. Other forms, almost round and clearly encysted, were fairly abundant in blood obtained by liver puncture. Sometimes very small parasites occurred in the erythrocytes; exceptionally they occurred in leucocytes (probably phagocytosed).

Two parasites with flagella have been seen. The parasite has not been cultivated, and the author is awaiting the results of subinoculations.

Sections of the tumefaction have shown typical granulomatous tissue, some elements resembling the parasites.

Franchini is convinced, from numerous examinations, that this parasite cannot be identified with the known haematozoa. It behaves like a protozoon and more especially a flagellate. The name *Haemocystozoon brasiliense* is proposed for it.

The preparations have been examined by Professor LAVERAN, who considers that the organism is a new Haemoprotozoon.

ii. In the second paper, Franchini in collaboration with Mantovani describes the case at length and gives a coloured plate of 70 figures illustrating the parasite. There is little or nothing of essential importance in this longer account which is not set forth succinctly in the first paper. It may be noted that no pigmented parasites are figured.

H. B. F.

DE RAADT (O. L. E.). Ueber einen bisher unbekannten menschlichen Krankheitserreger. [A New Pathogenic Organism in Man.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Mar. 15. Vol. 68. No. 3-4. pp. 318-322. With 1 plate and 1 chart.

De Raadt believes that he has discovered a new excitant of human disease in small bodies found in the spleen of a patient in the lazaret of Long-Iram, Central Borneo. The patient was a soldier aged 44, born in Java (a Madurese). There was a previous history of malaria, the species not being stated. When he entered hospital, he had fever that set in without previous shivering. Blood examinations were negative for malaria. Splenic enlargement was present, but the liver was not palpable. The diagnosis varied between malaria tropica and typhoid. A temperature chart is given, together with the therapy in full of the case, the main feature of which was great emaciation and variability in the size of the spleen. Details of the post mortem made immediately after death are given. The most noticeable points were:—

The lungs were heavily pigmented. The spleen weighed 200 grams and on its capsule being cut the pulp gushed out. The liver weighed 1,350 grams. It showed no cirrhosis. The conclusion from the post-mortem was that an infectious disease was highly probable.

Seven spleen smears had been made. They were stained with Giemsa from 1 to 24 hours and some were restained with Heidenhain's haematoxylin. The author details the results under four heads:—

(1) *Form, structure and size.* The organisms are usually ring-like with a large vacuole. The alveolar protoplasm collects at one side of the vacuole. No nucleus is present as such, though scattered granules of chromatin appear in some on using Heidenhain's stain. Pear shaped forms, also enucleate, occur. The structures vary from the most minute dimensions to  $3\mu$ .

(2) *Multiplication.* This is two-fold: (1) by budding, (2) by fission into two.

(3) *Occurrence in definite body cells.* They are rarely intraglobular, but are often within the large mononuclear leucocytes, without, however, showing intraphagocytic degeneration. Sometimes they are embedded in a matrix similar to that derived from the bursting of leucocytes. They are often quite free in the blood plasma.

(4) *Staining capacity.* Giemsa colours the protoplasm blue; there is no chromatin but violet dots may appear with intensive

staining. Heidenhain (after Giemsa) gives a grey-brown alveolar protoplasm, often with dark points or granules, but no individualised nucleus can be seen.

The author concludes that the minute bodies thus described are micro-organisms belonging to the Protozoa and probably related to *Babesia*. Von PROWAZEK, who saw the structures, first suggested the possibility of yeasts, but later considered they might be Protozoa of a new class. SWELLENGREBEL, who also saw them, considered them Protozoa on the ground of THEILER and BALFOUR's work on *Anaplasma marginale*. ROCHA-LIMA tested the preparations from the fungus point of view and pronounced them Gram-fast. The author concludes by stating that on the ground of their very typical structure he has named these organisms *Ovoplasma anucleatum*.

He has now found the same structures in spleen smears of two other cases from Java. He thinks there is a possibility that the parasite in many cases may be relatively harmless, but under certain conditions may become pathogenic.

The coloured plate contains 17 figures, all showing the bodies.

H. B. F.

PATTON (W. S.). *Studies on the Flagellates of the Genera Herpetomonas, Crithidia, and Rhynchoidomonas. No. I. The Morphology and Life History of Herpetomonas culicis*, Novy, MacNeal, and Torrey.—*Sci. Mem. by Officers of the Med. & Sanit. Depts., Govt. of India*. 1912. New Ser. No. 57. pp. 1-21. With 1 plate and 2 text-figures. Calcutta: Supt. Govt. Printing, India.

The author first defines the scope of the researches on *Herpetomonas*, *Crithidia* and *Rhynchoidomonas*. He points out that his views regarding the nature of the *Herpetomonas* of house-flies differ from those of certain other observers because he has worked with bred flies which have been infected experimentally while other workers have used flies caught at large. Consequently while he was able to follow the entire development of the flagellates and also to prevent mixed infections of flagellates such as occur in many insects, such a course is impossible when flies caught at large are used. Confusion of the flagellates of insects with pathogenic forms is easy unless the life-history of the parasites of the insect suspected of transmitting disease is known.

The flagellates of mosquitoes are then considered, and a detailed historical review of the records of *Herpetomonas* and *Crithidia* in the guts of mosquitoes is given. There is a great variation in the percentage of female mosquitoes that contain flagellates, depending largely on where the mosquitoes were taken. Mosquitoes collected from wells are nearly always infected with flagellates. Those bred from larvae in a tank are never infected—a point of importance in feeding experiments.

Patton points out that neither the parasite of Indian nor Italian kala azar will flagellate in any medium containing bacteria. Hence it is likely that FRANCHINI has encountered herpetomonads

natural to mosquitoes and not developmental forms of kala azar parasites.

The author details fully his methods of collecting and breeding the mosquitoes, and the technique employed in examination. *Culex fatigans*, *C. concolor* and *Joblotia* sp. all were breeding in the same tank. The first named only was ever found infected, pointing to the *Culex fatigans* being the specific host in Madras.

The preflagellate stages of *H. culicis* occur chiefly in the larval gut, occasionally in the gut of the nymph or imago. They are massed together just posterior to the junction of the Malpighian tubes with the midgut. Early forms are about  $4\mu$  in diameter and globular, and have vacuolated protoplasm with nuclei either central or to one side. A flagellar myoneme arises close to the blepharoplast. Binary fission occurs. No intracellular stage was ever seen. The multiplicative stage occurs in the gut of the larva but can also be found in the nymph or imago. Patton's experiments show clearly that the food of the insect has a direct bearing on the life of its *Herpetomonas*, for if the larvae are starved by being kept in clean water, the development of the flagellate is retarded and the mature insects hatch out still containing the preflagellate stages of the parasite, which have a great resemblance to the parasites of kala azar as liberated from leucocytes. There are, then, grave risks of misinterpretation should such insects be used for feeding experiments.

The growth of flagella in the preflagellate stages usually occurs in the larval gut just as the larva is about to become a nymph. They may flagellate singly or form rosettes. In rosette formation, nucleus and blepharoplast divide repeatedly and the daughter blepharoplasts are directed towards the centre. The mature flagellate, best seen in the gut of the imago as it emerges from the nymph, is from  $15\mu$  to  $25\mu$  by  $3\mu$  to  $5\mu$ . The flagellum may be  $25\mu$  to  $40\mu$  long.

The effect of different foods on the flagellates was tried. Banana supplied to larvae caused the flagellates to disappear, as the fruit developed yeasts that killed the flagellates. Meals of human blood killed the flagellates in practically every case. A similar result occurs when bugs ingest human blood and the fresh blood comes in contact with the flagellates of kala azar. These experiments probably give the clue as to how a harmless flagellate may evolve into a pathogenic one.

The post-flagellate stages are most easily studied in the male mosquito, which will live in captivity for a short time with little or no food. The flagellates fix themselves by their flagellar ends to the intestinal wall and divide there very regularly, so that palisades of them occur. The post-flagellates adhere together and are then passed out en masse with the faeces.

The author concludes that *H. culicis* is a natural parasite of *Culex fatigans* and is in no way connected with any blood parasite. It and many of its allied forms must be rigorously excluded in any experiments carried out with mosquitoes and the parasites of kala azar and oriental sore.

H. B. F.



CHATTON (Edouard) & LÉGER (Marcel). *L'Autonomie des Trypanosomes propres aux Muscides démontrée par les Elevages purs Indéfinis.*—*Compt. Rend. Soc. Biol.* 1913. Mar. 14. Vol. 74. No. 10. pp. 549-551.

The authors first review briefly some of the literature relating to the flagellates of flies from 1908 when CHATTON and ALILAIRE announced the coexistence of a trypanosome and a *Leptomonas* in a *Drosophilid* fly. They refer next to the announcement of Chatton and A. LÉGER (1911) of the dualistic hypothesis regarding the "eutrypanosomes" of the Malpighian tubules and the trypanosome-forms (leptotrypanosomes or trypanoïdes) of the intestinal *Leptomonas*. They refer also to the fact that they obtained pure strains of *Leptomonas* and of trypanosomes [see this *Bulletin*, Vol. 1, p. 499]. These pure strains have been maintained.

The pure trypanosomes, found in 80 per cent. of the infected flies, have been kept for two years and flies containing trypanosomes have been given to other workers who have been able to follow them up in their own laboratories. They have never shown *Leptomonas*.

Similarly the *Leptomonas* maintained for one year, though less successfully, has never contained trypanosomes. The difference of success in raising *Leptomonas* and trypanosomes is considered to be due to the varying chemical composition of the intestinal medium compared with that of the Malpighian tubules.

The authors consider that the specific autonomy of the trypanosome of the *Drosophilae* is definitely fixed. They also consider that "this demonstration holds for all flagellates of the same type described in *Muscidae*." [Surely this is generalising far too widely from a single particular case.]

In conclusion, the discovery by one of the authors of a Malpighian trypanosome in the house flies of Bastia, Corsica, is announced. As its cysts differ clearly from those of *T. drosophilae*, *T. luciliae* of PATTON [PATTON did not describe the parasite as a trypanosome] and *T. intestinalis* of ROUBAUD, it is designated by the author as *T. muscae domesticae*.

H. B. F.

CHATTON (Edouard). *Position Systématique et Signification Phylogénique des Trypanosomes Malpighiens des Muscides. Le Genre Rhynchoidomonas Patton.*—*Compt. Rend. Soc. Biol.* 1913. Mar. 14. Vol. 74. No. 10. pp. 551-553.

PATTON in 1910 stated that certain flagellates which he found in the Malpighian tubes of *Lucilia serenissima* were autonomous and belonged to a new genus, *Rhynchoidomonas*. ROUBAUD, working on allied forms, also considered them autonomous and placed them in the sub-genus *Cystotrypanosoma* [see *Sleeping Sickness Bulletin*, Vol. 4, p. 191]. ALEXEIEFF recently placed them in the genus *Crithidia*.

PATTON defined *Rhynchoidomonas* by the absence of free flagellum, postnuclear position of the blepharoplast, and lengthening of the posterior extremity of the body, all characters

occurring, according to Chatton, in sanguicolous trypanosomes. Thus defined, Chatton considers *Rhynchoidomonas* a synonym for *Trypanosoma*. Malpighian trypanosomes of the type *T. drosophilae* encyst by flexion of the body into a U, while *Crithidia*, *Leptomonas*, *Herpetomonas* and *T. grayi* encyst by contraction of the body. In fission, *T. drosophilae* begins to divide at the posterior pole, while the sanguicolous trypanosomes, *Crithidia*, and *T. grayi* begin by the anterior pole. *T. drosophilae* has its blepharoplast constantly postnuclear; *Crithidia*, *T. grayi* and cultural and evolutionary forms of sanguicolous trypanosomes have ante-nuclear blepharoplasts. Chatton also discusses the ethological characters of these flagellates. He considers it necessary to re-institute the genus *Rhynchoidomonas* for the Malpighian trypanosomes, characterised thus:—

Encystment by flexion, posterior fission, blepharoplast constantly post-nuclear; parasites specialised to Muscidae. Type of genus: *Rhynchoidomonas drosophilae* (Chatton et Alilaire, 1908).

This definition excludes *T. grayi*, which only differs from a typical *Crithidia* by the frequency of forms with a post-nuclear blepharoplast.

H. B. F.

RODHAIN (J.). **A propos de *Leptomonas pangoniæ* et *Trypanosoma denysi*. Note Rectificative.**—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 181-182.

While in Africa Rodhain and his colleagues described, under the name *Leptomonas pangoniæ*, a parasitic flagellate from the intestinal tract of *Pangonia infusca* in Bas-Katanga (this *Bulletin*, Vol. 1, p. 154). On returning to Europe they find that the flagellate must be placed in the genus *Crithidia* of PATTON and STRICKLAND. Hence the name of the parasite is *Crithidia pangoniæ*.

Further, in October 1912 they described a flagellate, *Trypanosoma denysi*, from the blood of the flying squirrel of Katanga. While in Africa the squirrel was identified as *Pteromys volant* inaccurately; the host is really *Anomalurus fraseri*.

H. B. F.

RODHAIN (J.), PONS (C.), VANDENBRANDEN (F.), & BEQUAERT (J.). **Note sur des Trypanosomides intestinaux d'*Haematopota* au Congo Belge.**—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 182-184. With 1 text-figure.

The paper contains a short account of two flagellates parasitic in the alimentary tracts of *Haematopota duttoni* and *H. vandenbrandeni* respectively. The parasite of *Haematopota duttoni*, found at Kasongo, was present in the rectum of two female flies out of eleven examined. Flagellated and cystic forms were present. The mobile parasites, large and stumpy, resemble fairly those figured by BRUCE, HAMERTON, and BATEMAN as occurring in *Tabanus secedens*. [It should be remembered that

these parasites were the *Crithidia tabani*, described prior to BRUCE by PATTON.] Pyriform or oval cysts corresponded to the "spermoides" of CHATTON.

At Sankisia another flagellate was found in *Haematopota vandenbrandeni*; 39 females were dissected, but only one was parasitised. Intestinal smears showed flagellated forms and cysts.

The flagellates had the form of a long narrow *Crithidia* with a pointed posterior extremity and narrow undulating membrane. The largest forms are from  $45\mu$  to  $50\mu$  long, with a body length of  $28\mu$  to  $30\mu$  and free flagella of  $16\mu$  to  $18\mu$ . The mean breadth at the level of the nucleus is  $1.2\mu$  and does not exceed  $2\mu$ . The nucleus is  $10\mu$  to  $14\mu$  from the anterior end of the body, possesses no karyosome but has scattered chromatic granules. Its dimensions are  $2.18\mu$  by  $1.2\mu$ .

The punctiform blepharoplast is  $2\mu$  to  $4\mu$  in front of the nucleus, and a relatively thick flagellum abuts on it. The cytoplasm of the posterior end has metachromatic granules frequently. The anterior part gradually merges into the free flagellum, the undulating membrane lying close to the body.

Smaller flagellates, only  $31\mu$  to  $39\mu$ , of which  $9\mu$  to  $16\mu$  forms the free flagellum, and having a breadth not exceeding  $1\mu$ , also occur. Between the flagellates and the encysted stages no intermediate forms occurred.

The cysts are surrounded by a thin, eosinophilic coat. They are ovoid, measuring  $5\mu$  to  $6\mu$  by  $2\mu$ , and the nucleus and blepharoplast are contained in the wider part of the cyst. Together with these cysts, aflagellar forms occur which the authors believe to be the first stages of condensation prior to encystment. The shortened, club-like bodies are  $14\mu$  by  $4\mu$  at the base, have deep staining protoplasm, and nucleus and blepharoplast very near each other.

The authors name this flagellate *Crithidia tenuis* and consider that it is probably a parasite peculiar to *Haematopota vandenbrandeni*.

The text figure illustrates various stages of *C. tenuis*.

H. B. F.

FANTHAM (H. B.). Note on the Specific Name of the *Herpetomonas* found in the Dog-flea, *Ctenocephalus canis*.—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 254-255.

The author points out that he named the *Herpetomonas* found in the gut of the dog-flea *H. ctenocephali* in July last. He briefly reviews the literature relating to the flagellate and remarks that the specific name *H. pseudoleishmania* proposed by BRUMPT (see this *Bulletin* Vol. 1, p. 669, footnote) cannot stand.

W. YORKE.

LAVERAN (A.) & MARULLAZ (M.). Au Sujet des Toxoplasmes du Lapin et du Gondi.—*Compt. Rend. Acad. Sciences.* 1913. Mar. 25. Vol. 156. No. 12. pp. 933-936.

The authors briefly survey the literature relating to *Toxoplasma*, which was discovered by A. SPLENDRE (1908) in an

epizootic among rabbits in Brazil. A similar parasite was recorded shortly afterwards by NICOLLE and MANCEAUX from the gondi (*Otenodactylus gondii*), a rodent allied to the guinea-pig, occurring in Tunisia.

The present work was undertaken to try to establish the identity or otherwise of *Toxoplasma cuniculi* of the rabbit and *T. gondii*. There are very great morphological similarities between the two parasites, strongly favouring their identity. Biological characters might show distinctions; hence they were tried, chiefly from the point of view of inoculability to the same species of animals.

*T. cuniculi* has been successfully inoculated to the rabbit, guinea-pig, pigeon, several Passerine birds, *Euphonia*, and unsuccessfully to the white rat.

*T. gondii* has been successfully inoculated to the gondi, guinea-pig, mouse, pigeon and Java sparrow, and unsuccessfully to *Macacus*, dog, white rat and rabbit.

Laveran and Marullaz investigated the inoculability of *T. gondii* (sent by C. NICOLLE) and find a difference regarding the reaction of the virus in the rabbit from what was found by C. NICOLLE and CONOR. Intraperitoneal inoculation of rabbits only succeeded with great difficulty (one case out of three and in that, a young rabbit, the parasites were very rare). An intravenous inoculation of one young rabbit succeeded easily, producing general infection with numerous free and dividing parasites in the internal organs, especially in the spleen and liver.

Laveran and Marullaz consider their experiments show that *T. gondii* can produce an infection in the rabbit (especially in young animals) comparable with that produced by *T. cuniculi*. Both are inoculable to pigeons. Finally they mention that a natural toxoplasmosis of the rabbit has been observed by G. BOURRET at St. Louis, Senegal. The authors in conclusion state that without wishing to assert definitely the identity of *T. cuniculi* and *T. gondii*, yet the arguments in favour of such identity appear to possess considerable weight.

H. B. F.

NICOLLE (Charles) & CONOR (Marthe). **La Toxoplasmose du Gondi. Maladie naturelle. Maladie expérimentale.**—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 160-165.

LAVERAN (A.) & NATTAN-LARRIER. **Au Sujet des Altérations anatomiques produites par le *Toxoplasma cuniculi*.**—*Ibid.* Mar. No. 3. pp. 158-160.

LAVERAN (A.) & MARULLAZ (M.). **Infections du Lapin par le *Toxoplasma gondii*.**—*Ibid.* Apr. No. 4. pp. 249-254.

Nicolle and Conor describe the evolution of *T. gondii* in the gondi, mouse, guinea-pig and pigeon. There are indications of a seasonal periodicity of infection. The number of parasites in the organs varies, the spleen, liver and lungs, kidneys, blood and bone marrow being successively less parasitised. Multiplicative forms of the parasite were numerous. The cysts of

SPLENDORE are considered as agglomerations by the present authors. By experiments they have established that the toxoplasma of the gondi can be maintained in laboratories in mice and pigeons. In mice and guinea-pigs the infection is local, in the pigeon it is generalised. Nicolle and Conor consider *T. gondii* is different from *T. cuniculi*.

Laveran and Nattan-Larrier have examined the anatomical alterations produced by toxoplasms in the liver, spleen and lungs of rabbits. The liver showed numerous necrotic nodules, varying in size up to 1 mm. in diameter. The infected hepatic cells usually contained 8 or 10 parasites, while 20 to 30 were sometimes present. The spleen showed much congestion and ochreous pigment was present. The lesions were less systematised than in the liver. The lungs showed nodular lesions, accompanied by oedema and congestion. Full details of the structure of the necrotic areas of each organ are given.

Laveran and Marullaz in the present paper give details of their experiments with *T. gondii* in rabbits, the main results of which are given in their paper reviewed above. They suggest that toxoplasms secrete an irritant toxin producing necrosis. The parasitised animals become thin and anaemic.

H. B. F.

FLU (P. C.). Over een Prowazekia-vorm (*Prowazekia Javanense*) in de ontlasting van een Patient te Weltevreden. [On a Species of *Prowazekia* (*P. javanense*) in the Faeces of a Patient from Weltevreden.]—*Geneesk. Tijdschr. v. Neder.-Indië*. 1912. Vol. 52. No. 6. pp. 659-678. With 1 plate.

The author investigated agar cultures of a flagellate obtained from the faeces of a patient from Weltevreden, Java. The flagellates grew abundantly on all kinds of nutrient media, but best of all on ordinary, slightly alkaline, nutrient agar, and remained alive for fourteen days, and on one occasion as long as three weeks. In older cultures cysts were found as well as motile forms. The optimum temperature for their growth appeared to be 26° C. The parasites were examined in both fresh and stained preparations. The organisms are 12 $\mu$  long and 5 $\mu$  broad, showing an oblong, broad lancet shape. There are also shorter and stouter forms, which are pear-shaped and on one occasion small, almost round parasites were seen. The flagella are anterior and lateral respectively, but the lateral flagellum is attached for a short distance to the cell body, becoming free posteriorly.

A cytological study of the flagellate was made. The central nucleus possesses a karyosome, and there is a large blepharoplast which is well seen in life. The two flagella arise from basal granules, near to which there is probably a small cytostome. Regarding the karyosome, the author states that the smaller the karyosome the more chromatin is deposited on the nuclear membrane. Apparently cyclic changes occur in the karyosome. Numerous food vacuoles may occur in the posterior part of the

body. Multiplication takes place by longitudinal fission. Details of encystment are given. The occurrence of autogamy is uncertain.

Regarding the question of the species of *Prowazekia* described in the paper, the author compares it with *P. cruzi*, *P. asiatica* and *P. weinbergi*. He proposes to name his species *Prowazekia jaranense*, but states that the name is merely a temporary one, as in course of time it may be shown that there is only one species of *Prowazekia*, as believed by Doflein. The author thinks that perhaps the species *asiatica*, *weinbergi*, and *jaranense* will ultimately prove to be the same and then the parasite described by him will be called *Prowazekia asiatica* (Castellani and Chalmers).

The paper is illustrated by a plate of 34 figures.

H. B. F.

CHATTON (E.) & ROUBAUD (E.). Sporogonie d'une Hémogrégarine chez une Tsétsé (*Glossina palpalis* R. Desv.)—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 226-233. With 2 plates.

The authors describe what they consider to be sporogony of a haemogregarine in *Glossina palpalis*. The flies were captured at Kolda, on the banks of the Casamance. Infected flies occurred only in this neighbourhood. Four were infected out of 465. The parasites were found in the body cavity of the flies. When examined fresh, the presence of the cover-glass was sufficient to cause the bursting of the cysts, issue of sporocysts and subsequently of motile sporozoites. Stages corresponding to copulation were lacking. The youngest forms were sub-spherical, voluminous, uninucleate,  $50\mu$  to  $75\mu$ . A thin membrane was present. The nucleus showed a central karyosome. Next a dozen large, plumose chromosomes replaced the karyosome. The authors think that if the subsequent stages were known they would resemble those of certain Gregarines and Coccidia. Each chromosome becomes a daughter nucleus, and the daughter nuclei themselves multiply by a division more of the Coccidian than of the Gregarine type. The nuclei at first are distributed but later collect at the periphery, where ultimately a series of uninucleate prominences are produced. Each prominence or bud ultimately detaches itself, and its volume doubles as it does so, whereby it becomes  $10\mu$  to  $12\mu$  in diameter. These buds or sporoblasts use the central residuum of their parent as food and when this is exhausted they measure  $20\mu$  to  $25\mu$ . Sporozoite formation follows. The binucleate stage of the sporoblast is somewhat prolonged but succeeding divisions are quicker. It is again noted that the divisions are of the Coccidian type and analogous with those of *C. schubergi* and *Adelea zonata*. The nuclei become arranged in two parallel planes. The lines of separation are at right angles to these planes. The sporocyst contains as many sporozoites as there were nuclei, i.e., more than 20. The

cysts contain a hundred sporocysts. Isolated vermicular sporozoites when stained with Giemsa show a red nucleus at the posterior third of the body and on either side of it an area staining intensely violet.

The affinities of the parasite are discussed. The authors arrive at the conclusion that it is part of the cycle of a haemogregarine, either mammalian or reptilian. Finally they think that this parasite of *Glossina palpalis* is the sporogonic stage of a Haemogregarine of a reptile, such as Varanus, Crocodilus or a lizard.

The paper is well illustrated.

[Are the authors sure that the organism examined is not a natural parasite of *Glossina palpalis*?]

H. B. F.

LEGER (André). Parasite des Hématies, Genre *Grahamella* (Brumpt), de *Mus maurus*, (Gray).—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 247-249.

Léger reviews the previous accounts of *Grahamella*, first discovered by GRAHAM-SMITH in 1905 in the erythrocytes of the mole, found by THOMSON in 1906 in the same animal, by BALFOUR in the jerboa, by FRANÇA in the dormouse and field mouse, and by BRUMPT in 1911 in mole's blood again. BRUMPT considered the rod-like structures to be true protozoa and founded the genus *Grahamella* for their reception. Léger has now found similar structures in the erythrocytes of *Mus maurus*, the rat of Bamako, Haut-Sénégal-Niger. He found them in 28 animals out of 125 examined. The number of bodies in each erythrocyte varied between 30 and 80. Infection of peripheral blood was poor, that of heart blood relatively heavier.

Attempts at the experimental infection of other *Mus maurus*, guinea-pigs and rabbits have failed, whether intravenous, intraperitoneal or subcutaneous inoculation were employed.

H. B. F.

NEGRI (Adelchi). Beobachtungen über *Haemoproteus*. — *Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Apr. 23. Vol. 68. No. 7. pp. 599-602. With 1 coloured plate.

The author reviews the previous work of SCHAUDINN on *Haemoproteus* (*Halteridium*) *noctuae* and of ARAGAO on *H. columbae*. He worked more especially on the latter parasite. He obtained his infected doves from the Roman Campagna, and found all the forms which have been described from the peripheral blood and from the leucocytes in the lungs. He confirms ARAGAO's work thereon.

No *Lynchia* were found on the doves, so that the author could not investigate the stages in the invertebrate host. Although he had abundant infected material he was unable to confirm SCHAUDINN's statements regarding a developmental relationship between *Halteridium* and *Trypanosoma*. No intermediate forms between these types were seen, nor were flagellates of any kind obtained in blood cultures.

The paper is illustrated by a coloured plate of four figures, showing the minute merozoite forms of *H. columbae* in the lungs.

H. B. F.

AWERINZEW (S.). *Ergebnisse der Untersuchungen über parasitische Protozoen der tropischen Region Afrikas. I.* [Researches on the Parasitic Protozoa of Tropical Africa. I.]--*Zoologischer Anzeiger*. 1913. Jan. 3. Vol. 41. No. 4. pp. 186-188.

The author states that this paper is the first of a series of preliminary communications on his investigations at Daressalam, Amani, Zanzibar, Delagoa Bay, Durban and other places in East Africa. The present paper relates to a protozoan parasite from the blood of the frog, *Rana nutti*, the host being found in the neighbourhood of the Imperial Biological and Agricultural Institute at Amani, German East Africa.

The parasites occurring in the red blood corpuscles of *Rana nutti* at first were considered as true haemogregarines. Examination of stained preparations led the author to place them in the Binucleata, apparently as descendants from trypanosome-like stocks.

The parasites in the red blood corpuscles as well as those in the plasma show two tiny bodies in their broad stumpy ends. These two small bodies take up nuclear stains. The author emphasises that the occurrence of these granules is the general rule. In a few cases he has been able to find a connection between one of these small bodies and the nucleus, and figures one example of this. He has never seen a vestige of a flagellum or undulating membrane in these parasites and, in his opinion, the gregarine-like blood parasites can be placed either with the Coccidia or with the Trypanosomes, as well as with the Piroplasmidae and Plasmodiidae.

A new generic name is necessary to avoid confusion arising from the two-fold origin (and therefore also two-fold structure) of these organisms known as haemogregarines.

In a few cases some of the free swimming forms in the blood of *Rana nutti* showed a long ribbon-like nucleus resembling what PROWAZEK described in the Haemogregarines of the gecko, *Platydictylus guttatus*, but filling nearly the entire cell. The plastin takes no share in this nuclear growth. In some frogs only forms rich in chromatin occurred and then in small numbers. In other cases along with erythrocytic parasites there were masses of free haemogregarine-like forms of which a few had enlarged nuclei, but the greater number possessed a small compact nucleus, near which a few chromatin lumps lay sometimes. Those parasites, which show increase of nuclear chromatin, were two to three times as large as the others. Whether male, female and indifferent forms were present is undecided. In conclusion it is stated that apparently sexual processes as well as ordinary multiplication take place in the body of the frog, but no further information is given regarding these points.

The paper is illustrated by four text figures.

H. B. F.



VIGUIER (G.) & WEBER (A.). i. Les Mitochondries de l' *Haemogregarina sergentium* durant son Evolution dans le Sang du Gongyle.—*Compt. Rend. Soc. Biol.* 1913. Apr. 11. Vol. 74. No. 12. pp. 664-666.

ii. Nouvelles Observations sur l'Altération des Hématies sous l'Influence d'une Hémogrégarine chez le Gongyle. — *Ibid.* Apr. 18. No. 13. pp. 760-761.

i. The authors have studied the mitochondria\* of *Haemogregarina sergentium* of the lizard, *Gongylus* sp., from the time of the penetration of the merozoite into the erythrocyte until the destruction of the host cell. Free merozoites are  $3\mu$  to  $6\mu$  long. They each contain fine chromatic aggregations resembling a karyosome, whose extremities are fairly often covered by two semilunar masses where one or two mitochondrial granulations occur. At the time of penetration the merozoite elongates and the mitochondria, which are fine, are in about a dozen groups or are irregularly distributed. Some suggest multiplicative stages. One group of mitochondria is always near the nucleus, another at one extremity. Vesicles, which stain like mitochondria and perhaps may result from their alteration, may be present.

At a certain stage, the mitochondria which are then numerous become scattered throughout the cytoplasm. In addition grains staining like the nucleus make their appearance (extra-nuclear chromatin or chromidial apparatus). As the haemogregarine grows, the mitochondria arrange themselves at the periphery, and simultaneously the capsule of the haemogregarine appears, and these phenomena may not be unrelated.

In still older organisms, the mitochondria become few and very scattered, while in haemogregarines that seem degenerated within the red cells the mitochondria have disappeared.

ii. In the second note the authors briefly refer to the alterations of the erythrocytes by the *Haemogregarine*, as seen in the blood of one reptile (*Gongylus*) out of 13 parasitised, the red cells of the others exhibiting no particular alterations. In the case discussed, fragmentation of the nuclei of the red cells occurred. Mitochondria were also demonstrated by special methods (those of REGAUD)

H. B. F.

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\* "Amongst the granulations of the protoplasm, special mention must be made of the bodies known generally as *chondriosomes* and *mitochondria* . . . . The chondriosomes are not to be classed with the temporary, metaplastic inclusions, but are permanent elements of the cell-protoplasm. The chondriosomes of Protozoa have recently been the subject of detailed study by Fauré-Fremiet. In the living condition they are small transparent bodies, feebly refractile, and of a pale grey tint. In shape they are generally spherical, and vary from  $0.5\mu$  to  $1.5\mu$  in diameter . . . . When the nucleus (in Infusoria the micronucleus) divides, the chondriosomes also divide simultaneously, and the daughter-chondriosomes are sorted out between the two daughter-cells; they have, however, no direct relation with the nuclear apparatus . . . . Fauré-Fremiet considers that they "play an important part in the life and evolution of the sexual cell," in Protozoa or Metazoa." (MINCHIN (E. A.). *An Introduction to the Study of the Protozoa with Special Reference to the Parasitic Forms.* 1912. London: Edward Arnold. p 41.)

FLU (P. C.). Over de z.g.n. Kurlofflichamen in de mononukleaire Bloedcellen van *Cavia cobaya*. I. Morphologische Studiën. [On the so-called Kurloff Bodies in the Mononuclear Blood-Cells of *Cavia cobaya*.]—*Geneesk. Tijdschr. v. Neder.-Indië*. 1912. Vol. 52. No. 6. pp. 679-702. With 1 coloured plate.

The author reviews the previous work done on the subject and discusses the different opinions held by various investigators as to the nature of the Kurloff bodies. He refers to his previous work with PAPPENHEIM in Berlin. The investigations here recorded were done in Weltevreden (Java) where 100 per cent. of the guinea-pigs examined were found to be infected and 25 to 40 per cent. of their monocytes contained Kurloff bodies, while only 30 per cent. of the animals were infected in Berlin and 10 to 12 per cent. of the monocytes contained the bodies.

The bodies were examined intra vitam and by various methods of fixation and staining.

The author concludes:

(1) That the Kurloff bodies possess movements of their own.

(2) That the movement is an amoeboid one and is performed by the peripheral as well as by the central parts of the body; hence it is improbable that the poorly staining mass of the Kurloff bodies, termed by the author 'ground substance,' is a reaction-product of the mononuclears.

(3)-That by staining Kurloff bodies fixed in sublimate solution with Loeffler's methylene blue, morphological appearances are obtained which correspond closely with those of the so-called Chlamydozoa.

The paper is illustrated by a coloured plate, containing 39 figures.

H. B. F.

PONSELLE (A.). Culture *in vitro* du *Trypanoplasma varium* Leger.—*Compt. Rend. Soc. Biol.* 1913. Apr. 11. Vol. 74. No. 12. pp. 685-688.

Ponselle has succeeded in cultivating *Trypanoplasma varium*, parasitic in the blood of the loach, *Cobitis barbatula*, L. At first he obtained mixed cultures of *Trypanoplasma varium* and *Trypanosoma barbatulae*, both parasitic in the loach. Care was then taken to procure a droplet of cultural blood containing trypanoplasmas only, unmixed with trypanosomes. The cultures were made in a medium previously devised for the culture of trypanosomes (see this *Bulletin* Vol. 1, p. 676 and Erratum below), using also a modified Petri dish known as a "boîte de Bordas" for hanging drop cultures. Two passages from tube to tube have now succeeded. The cultural forms of the *Trypanoplasma* differ very little from the blood forms. The same disposition of nucleus, blepharoplast, and flagella and the same dimensions occur, while the movements are alike. The undulating membrane, however, is much reduced, and the posterior flagellum is free from the body for a greater or less distance, and so the cultural forms somewhat recall

*Prowazekia*. Division is always binary and longitudinal, nuclear division either preceding or accompanying that of the blepharoplast, as a rule.

No elongate, thin forms, similar to those occurring in the alimentary tracts of leeches feeding on infected fish, have been observed; nor have forms suggesting sexuality been seen.

The note is illustrated by 15 text-figures, and there is one figure of a cultural *Trypanosoma barbatulae* in addition.

H. B. F.

#### ERRATUM.

*Bulletin* Vol. 1, No. 11, p. 676. In the formula of PONSÉLLE'S modification of the N.N. medium, for 'unwashed gelatine' read 'unwashed agar.'

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## TROPICAL DISEASES BUREAU.

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## LEPROSY.

KUPFFER (A.). **Die Lepra in Estland.** [Leprosy in Esthonia.]—*Lepra*. 1913. Apr. Vol. 14. No. 1. pp. 14-32.

Leprosy was prevalent in Esthonia (the most northerly of the Russian Baltic provinces) in the Middle Ages, but apparently died out again in the 17th and 18th centuries. It had reappeared by the second decade of the 19th century, probably introduced by Cossacks from the South during the Napoleonic wars, but made little progress until the period succeeding the Crimean war; and Kupffer brings forward good arguments in support of his contention that the increased prevalence is due to the occupation of the province at that time by troops from infected districts of the South. Later opportunities for infection occurred through the return of natives after military service, and the increasing communications of the remoter districts with other parts of Russia. The total number of lepers recorded since 1828 is only 350, and at the present time there are outside the asylums only 30 known lepers among the 467,000 inhabitants of Esthonia. Since 1906 at least one third of all the lepers alive in Esthonia have been isolated in the official asylums, and about seven-tenths are so cared for at present. An account is given of the official regulations and the means adopted for the notification and control of cases as they occur.

J. Henderson Smith.

MEULENGRACHT (E.). **Communication concernant la Lèpre en Serbie.**—*Lepra*. 1913. Apr. Vol. 14. No. 1. pp. 33-34.

An account of a case of lepra mixta in a young man, whose father and elder brother suffered from the same disease. The interest of the case lies mainly in the fact that hitherto only 3 cases of leprosy have been recorded in Servia.

J. H. S.

SERRA (Alberto). *Lo Stato attuale della Lepra in Sardegna.* [Leprosy in Sardinia at the present time.]—28 pp. With 2 plates. 1912. Cagliari: Prem. Stab. Tipogr. Ditta G. DESSI.

A pamphlet giving the history of seven cases recently observed in Sardinia, and illustrating the much greater prevalence of leprosy than is officially recognised, and the necessity of energetic measures and constant watchfulness on the part of the officers of health.

J. H. S.

ZAMBACO. *La Lèpre dans l'Égypte actuelle.* — *Presse Méd. d'Égypte.* 1913. Mar. 15. Vol. 5. No. 6. pp. 89-92.

The number of cases of leprosy actually registered in Egypt is 2,204, but ENGEL, Director of the Statistical Department, estimates that there must be 8 or 9 times that number in the country. These cases mingle freely with the general population, follow every trade, and are in intimate contact with their families and neighbours. But no single instance of direct contagion has been conclusively established in Egypt, and no foreigner has contracted the disease in the country. Zambaco, laying emphasis on these facts, insists that it is impossible to look on leprosy as a contagious disease in Egypt, since every opportunity is given for the infection to spread, and without offering an alternative explanation of its persistence maintains that it is unscientific to uphold the contagion hypothesis in the absence of any direct evidence of its occurrence.

J. H. S.

EICHMULLER (G.). *Réflexions à propos de deux Cas de Lèpre observés à Tunis.*—*Lepra.* 1913. Apr. Vol. 14. No. 1. pp. 35-39.

Eichmuller records the occurrence of a case of ulcerative tuberculous leprosy, of which the mother was found to be suffering from the disease, while two cousins had already died from it. The connection between these four cases is not satisfactorily established, but Eichmuller considers that the mother was infected from the son, and that they are good evidence of the contagious nature of leprosy. He draws attention to the necessity of regulations for the control of leprosy in Tunis.

J. H. S.

McCoy (George W.). *Care of Lepers in Hawaii.*—*U.S. Public Health Rep.* 1913. Apr. 18. Vol. 28. No. 16. pp. 726-728.

There is a prevalent impression that the lepers in Hawaii are kept isolated on a barren island with armed guards and under conditions of extreme severity. As a matter of fact the leper establishment is situated on a small peninsula of the beautiful island of Molokai, there are no armed guards, and the patients are so contented as to show no wish to leave the settlement. The

disease was introduced some 60 years ago, probably by Chinese, and since 1864 a policy of segregation has been carried out. Anyone who knows of a case of leprosy in the islands is required to report it, and if on examination the suspect is found to be infected, he is committed to an isolation hospital at Honolulu, where he is treated for at least 6 months. If no improvement occurs in that time, he is removed to the Molokai settlement. There the patients are well cared for, marriage is allowed, children being removed to healthy surroundings, and ample food and clothing and medical attendance are provided. At present about 700 lepers are known to exist in the islands, 600 at Molokai and 100 in the isolation hospital at Honolulu, but there must be many more not reported. The number is decreasing, since the native Hawaiians who are the principal sufferers are becoming rapidly fewer. The Government is fully alive to the importance of the question, and the tax on the Hawaiian treasury for the care of lepers alone amounts to about 1 dollar per head of the entire population.

J. H. S.

## CLINICAL.

BROCQ, FERNET, & DELORT. *Lésions Ulcéreuses Gangréneuses et Phagédéniques chez un Lèpreux.*—*Bull. Soc. Française de Dermatol. et de Syphiligraphie.* 1913. Feb. Vol. 24. No. 2. pp. 57-69.

An account of a case of *lepra tuberosa*, which developed on the left leg several livid red patches which turned rapidly dark and eventually ulcerated. They became gangrenous, sloughed, and finally the ulcer that remained extended in the manner of phagedenic ulcers, which indeed they closely resembled. The process eventually ceased, and slow healing began. The condition is not unlike that which has been described as *lepre lazarine*, but the authors found no evidence to assure them that its occurrence was directly due to the leprosy bacillus.

[The subsequent discussion on this paper illustrates the difficulty of determining the prevalence of leprosy, since there was considerable difference of opinion as to whether 50 or 500 was nearer the actual number of lepers in the Department of Alpes-Maritimes.]

J. H. S.

CARRIEU (M.) & ANGLADA (J.). *Hépatite Lèpreuse à forme de Cirrhose Ascitique Biveineuse avec Présence de Bacilles de Hansen dans le Parenchyme Hépatique.*—*Arch. de Méd. Expérimentale et d'Anat. Pathologique.* 1913. Mar. Vol. 25. No. 2. pp. 149-167. With 4 text-figures.

The case, which is described in detail with illustrative sections of the liver, is one which had presented evidence of leprosy for a little more than 2 years, but came under the observation of the authors owing to marked signs of ascites that had been apparent for about 2½ months. This was not accompanied by any signs of peritonitis and was eventually shown to be due to extensive interstitial cirrhosis of the liver in both the portal and subhepatic areas,

producing a mechanical obstruction. Clinically there were quite definite signs of nodular and macular leprosy, but the bacilli could not be found in the nasal mucosa during life, nor were they found post mortem in any organ except the liver. There, however, they were present in enormous numbers typical in grouping, staining and other characters and not producing any lesions in guinea-pigs. Leprosy of the liver is a comparatively rare condition, usually met with only in general invasion of the viscera and accompanied by amyloid degeneration without a definitely specific local reaction. Occasionally a nodular form is met with or a diffuse interstitial change, which is more specific, but cirrhotic forms are rare, more particularly when the bacilli are present in large numbers. In this case there was undoubtedly some parenchymatous hepatitis, but the prominent change was the cirrhosis, which recalled the typical Laennec cirrhosis in its anatomical appearance and clinical effects. Nowhere else, except in the skin, was there any definite specific change; and the authors suggest that a tendency to alcoholism in the patient had determined the localisation of the lesions in the liver.

J. H. S.

## TREATMENT.

SERRA (Alberto). *L'Antileprol nella cura della Lepra*. [Antileprol in the treatment of Leprosy.] — *Gior. Ital. d. Malatt. Veneree e d. Pelle*. 1912. Vol. 53. (Anno 47.) No. 6. pp. 734-738. With 2 plates.

Serra has treated 10 cases with antileprol, a preparation derived from chaulmoogra oil, and is greatly impressed with its efficacy. It is so much better tolerated than the oil that it is possible to give high doses, and he has given as much as 10 grammes per day to one case by the mouth, 5 gm. twice daily subcutaneously to another case, and to a third 5 gm. subcutaneously and up to 4 gm. by the mouth, without producing any renal or alimentary disturbance. Four of the ten cases were treated for over 3 months and two for 6 months, and all six were greatly improved. They comprised 3 cases of lepra tuberosa, 1 of lepra mixta and 2 of lepra nervorum. Ulcers dried up and healed, lepromata were absorbed, laryngeal and nasal signs disappeared, thickening and swelling of arm and hands were reduced, bacilli lost their staining uniformity and many disappeared; even in the anaesthetic cases there was a considerable return of sensibility. Serra recognises that the improvement may be only temporary, but he looks on antileprol as the only specific remedy we have at present for the cure of leprosy.

J. H. S.

BROCQ & POMARET. *Nouveau Produit injectable pour le Traitement de la Lèpre*.—*Bull. Soc. Française de Dermatol. et de Syphiligraphie*. 1913. Feb. Vol. 24. No. 2. pp. 70-73.

JEANSELME (E.). *A propos de la Communication de MM. Brocq et Pomaret sur un Nouveau Produit injectable pour le Traitement de la Lèpre*.—*Ibid.* Mar. No. 3. pp. 149-150.

Oil of eucalyptus (5 or 10 per cent.) has been injected with benefit, it is said, in leprosy; and it was pointed out by Pomaret that

if 30 cc. be added to 70 cc. of chaulmoogra oil, which has been rendered fluid by warming, the mixture remains fluid at all temperatures, and can be bottled and sterilised ready for injecting. The mixture is tolerated much better than pure chaulmoogra oil. Jeanselme uses a mixture consisting of Chaulmoogra oil (washed in alcohol and filtered), 20 gr.; Guaiacol, 1 gr.; Camphor, 0.50 gr.; Vaseline oil pure, 20 gr.

J. H. S.

GAUCHER & BOINET. **Traitement de la Lèpre par les Injections Intraveineuses de Salvarsan et les Piqûres d'Abeilles.**—*Bull. Soc. Française de Dermatol. et de Syphiligraphie*. 1913. Mar. Vol. 24. No. 3. pp. 172-174.

A brief account of a case of lepra tuberosa with symptoms of 18 months' standing, which was treated daily for 40 days with bee-stings, in numbers which were gradually increased up to 150-200 (in all 3,935 were given), and also received 40 cgr. of Salvarsan. The result of this treatment seems to have been on the whole favourable, numerous ulcers on limbs, feet, hands &c. drying up and cicatrising quickly, and lepromata on the face, ears &c. atrophying but not disappearing completely.

J. H. S.

## EXPERIMENTAL.

SERRA (Alberto). **Dernières Recherches sur l'Inoculation de Matériel Lèpreux dans l'Oeil du Lapin.**—*Leprosy*. 1913. Feb. Vol. 13. No. 4. pp. 237-247.

Serra has already published the fact that he has isolated from developing lepromata a bacillus which corresponded completely to the anaerobic organism isolated by CAMPANA and later by DUCREY, and has given an account of his attempts to transmit the disease to animals. He found that the inoculation of the culture into the anterior chamber of the eye in rabbits was followed eventually by a nodular development indistinguishable from a leproma, while the serum of the animals developed the properties of agglutination and complement-fixation possessed by the serum of human lepers. Further, it was possible to produce nodules in fresh rabbits with material from animals previously injected. In this paper he reproduces a series of photographs illustrating the changes which occur in the anterior chamber after inoculation with normal skin, with young lepromata, with nodules from rabbits, and with culture material. No significant difference is apparent in the lesions of the series injected with culture or infected tissue, and multiplication of the organisms occurs; while in the animals injected with normal tissue the material injected eventually disappears. He has succeeded in isolating again from an experimental nodule the organisms injected, and with this bacillus has again produced nodules in fresh rabbits. He has not yet succeeded in isolating the bacillus from nodules produced by the injection of leproma tissue, a result which would go far to establishing the fact of the multiplication of the bacilli.

J. H. S.



MÖLLERS (B.). *Serologische Untersuchungen bei Leprösen.* [Serum-reactions in Leprosy.]—*Deut. Med. Wochenschr.* 1913. Mar. 27. Vol. 39. No. 13. pp. 595-596.

The capacity of leper-serum to fix complement in the presence of tubercle bacilli or of extracts made from them was tested with 32 sera. These came from various parts of the world (*e.g.* Japan, Egypt, Carpathia) to Berlin, and were not fresh; but the author found that the complement-fixing power, if present, remained intact for months, and even spontaneous deviation occurred only after 6-12 months' keeping in the Frigo apparatus. Of the 32 sera 20 were derived from cases of *lepra tuberosa*, and 19 or 95 per cent. of these gave positive results; 4 from *lepra mixta* with 100 per cent. positive results; and 8 from *lepra nervosa* with 25 per cent. positive. The negative tuberosa case was one which had been regarded as healed for 3 years and no longer gave a Wassermann reaction. Four preparations of tubercle were used in the experiments, viz. Old Tuberculin, New Tuberculin T. R. (Hoechst), Perlsucht Tuberculin (Hoechst), and a preparation of the bacilli similar to T. R. known as T. O. I. The two bacillary preparations gave better results than the others, which are culture-fluid preparations, and bovine tubercle appeared to give a more effective antigen than human. The author has little doubt that the high percentage of positive results points, not to a concomitant tuberculosis in the patients from whom the serum was derived, but to a group-reaction or relationship between the tubercle and the leprosy bacilli.

J. H. S.

GOUGEROT (H.). *Anaphylaxie Lèpreuse.*—*Lepre.* 1913. Feb. Vol. 13. No. 4. pp. 211-217.

Gougerot states that Rost's leprolin produces on injection into lepers a local and general reaction analogous to the tuberculin reaction and not obtained in normal individuals; and that it has a distinct curative action in fact that "treatment with leprolin is the best treatment we know of." There is a leprous anaphylaxis in the same sense as there is a tuberculous anaphylaxis, and he has twice demonstrated passive sensitisation of guinea-pigs by injecting them intraperitoneally with leper serum and afterwards injecting leprosy bacilli intracerebrally. Lepers will sometimes give a general reaction to tuberculin, and in two cases out of four he obtained an intense local reaction when the cuti-reaction was made with tuberculin over a leproma. The ophthalmo-reaction was always negative. Conversely, in cases of lupus and pityriasis rubra subcutaneous injection of 5 cc. of leprolin may produce a general reaction, although positive results were not obtained with ophthalmo- or cuti-reactions. He has twice out of 6 experiments demonstrated passive sensitisation by injecting tubercular antigen intracerebrally into guinea-pigs previously treated with leper serum, and 10 times out of 24 by injecting leprous antigen into guinea-pigs injected with tuberculous serum. Gougerot believes that the local anaphylactic state explains many difficult clinical problems, and our knowledge of its existence will help us in prophylaxis and treatment.

J. H. S.

## UNCLASSIFIED.

MONTESANTOS (Denis). **Un Coup d'Oeil sur la Question de la Contagiosité et l'Hérédité de la Lèpre.**—*Presse Méd. d'Egypte*. 1913. Apr. Vol. 5. No. 7. pp. 105-106.

The author in some speculative observations on this subject remarks firstly, that in many cases of leprosy there is a long period of latency, broken only by isolated and transitory symptoms, before the disease definitely declares itself, and this long interval frequently makes it impossible to trace the source of the infection; secondly, that the geographical distribution of leprosy has no relation to climatic or hygienic conditions; and thirdly, that among the less severe anaesthetic types of the disease cases occur which are really mild and may be looked on as attenuated forms of the disease. These latter, he asserts, occur only in regions where the disease has long been endemic, and are due to a partial immunity acquired by the remote descendants of lepers. There is thus a graduation in susceptibility to infection. He admits the occurrence of direct contagion, but considers that an individual who is attacked by the bacillus loses his power of resistance gradually during the long latent period, and this diminished resistance or increased susceptibility may also be transmitted to his descendants.

J. H. S.

BARBÉZIEUX (G.). **La Prophylaxie de la Lèpre au Tonkin. (Réponse à quelques Objections et Critiques.)**—*Bull. Soc. Méd.-Chirurg. de l'Indochine*. 1913. Mar. Vol. 4. No. 3. pp. 88-97.

Some remarks on the methods and objects of the system adopted for the treatment and care of lepers in Tonkin.

J. H. S.

THOMPSON (J. Ashburton). **Experimental Leprosy: a Perspective.**—*Leprosy*. 1913. Apr. Vol. 14. No. 1. pp. 1-13.

A review of some of the recent work on the bacteriology of leprosy, in which the identity of the organism isolated by KEDROWSKY with the chromogenic bacterium cultivated by DUVAL is taken as assured. [No reference is made to the work of BAYON on the subject.]

J. H. S.

FAVRE (Maurice) & SAVY (Paul). **Histologique Pathologie du Lépreux cutané aigu.**—*Arch. de Méd. Expérimentale et d'Anat. Pathologique*. 1913. Mar. Vol. 25. No. 2. pp. 225-232. With 1 plate.

A detailed description of the histological appearance of a nodule removed in the acute stage of development. The authors uphold the existence of a genuine lepra cell, which they regard as a connective tissue cell and of great importance in the formation of the nodule.

J. H. S.

## PLAGUE.

Low (R. Bruce). Preliminary Statement as to Occurrences of Plague and Cholera throughout the World during 1912.—*Reports to the Local Govt. Board on Public Health and Medical Subjects*. 1913. (New Series No. 78). pp. 22-33. (London: published by H.M. Stationery Office.)

This information was given in tabular form in Vol. 1, p. 557, of this *Bulletin*. In India there were more than 273,000 deaths from plague in the year 1912, compared with 842,000 in 1911. There is a population of 48 millions in the United Provinces of Agra and Oude; 158,074, 332,301, and 101,229 are the numbers of fatal cases recorded there in the years 1910, 1911, and 1912. The figures for the Punjab with a population of 24 millions are 169,867, 198,000, and 30,000 for those years. The Presidency of Bombay with a population of 27 millions had 100,399 deaths from plague in 1911, and about 32,000 in 1912. In Madras with its 46 million inhabitants, there were only 6,500 deaths from plague in 1912. Assam and the North West Frontier Province were almost entirely immune. During 1912, 1,708 deaths from plague occurred at the port of Bombay, 1,828 at Calcutta, 1,110 at Karachi, and 852 at Rangoon.

Hongkong suffered severely—1,729 deaths; there was an epizootic among rats at the same time. At Shanghai the rats were infected without an epidemic arising.

C. Birt.

## EPIDEMIOLOGY.

SWELLENGREBEL (N. H.). Mededeeling omtrent Onderzoekingen over de Biologie van Ratten en Vlooien en over andere Onderwerpen, die betrekking hebben op de Epidemiologie der Pest op Oost-Java. [Investigations on the Biology of Rats and Fleas, and on other Subjects relating to the Spread of Plague in East Java.]—*Geneesk. Tijdschr. v. Nederlandsch-Indië*. 1913. Vol. 53. No. 1. pp. 53-154. With 15 pages of charts and curves.

In the year 1910 plague broke out in the Province of Malang, a mountainous district, situated 400-1,000 metres above sea-level in the eastern part of the Island of Java. In a population of about three quarters of a million there were about 1,500 people attacked in 1911, and 2,240 in 1912. 753 cases have already occurred this year, and the disease has extended to the neighbouring provinces of Kediri and Madioen with a population of about 40,000 where over 1,200 attacks have been already reported, and it is still raging. A curve of plague incidence is given in which it is seen that there was a sudden rise in May, 1911, and a sudden fall in July and August; after remaining low from October, 1911,\* to April, 1912, it began to rise again and is still on the up grade. The climate is very uniform; the humidity does not fall below 70 per cent. of saturation; the temperature remains at 23°-25° C. throughout the year.

*Rats*.—The most widely distributed species of rat in Java is the *Mus rattus*, of which there are two varieties, the house-rat and the

field-rat; the former differs from the latter in its variable colour, in its tail being longer than the body, and in possessing 10 mammae instead of 12. There is a subvariety of the house-rat which is smaller and of more uniform colour, and has two pairs of inguinal mammae instead of three; it appears to be identical with *Mus concolor* described by LLOYD in Rangoon; in some parts as many as 45 per cent. of house rats are *M. concolor*. Of 615 rats captured in the boats and ships lying in the harbour of Soerabaia nearly all were *M. rattus* of the large subvariety; it has been found in goods wagons. The house rat migrates from house to house and from village to village. Breeding takes place throughout the year, but more gravid rats are found in April, May and December than in the rest of the year. From 1 to 12 per cent. of the rats caught in houses are field rats; they seek human dwellings when supplies in the country become exhausted.

*Mus decumanus* has a much more limited habitat than *M. rattus*, and occurs only at the seaports and along the river.

*Nesokia setifera*, *Crocidura coerulea*, and *Chiropodomys gliroides* and *C. anna*, though present, appear to play no part in the diffusion of plague in Java.

*Fleas*.—*Xenopsylla cheopis* and *Pygiopsylla ahalae* are the only rat-fleas found in the plague area. Some 20,000 flea counts have been done: in plague-free localities the average number of fleas per rat has ranged between 0.2 and 0.8. Many charts are given in which it is seen that as the average flea count increases so do the number of cases of human plague. A map of Malang also is included in which are shown the villages with rat flea counts above and below 1.7, and plague distribution; the plague areas correspond with the high flea ratio.

The eggs of *X. cheopis* hatch in 4-8 days, and the larvae develop into mature fleas in 31-52 days. Humidity is favourable to their development, for when the air was driest fewer eggs hatched, and the larval and pupal stages were prolonged; too much moisture, however, kills the larvae in 9 days.

Of 38 fleas reared in the laboratory 36 survived 7 days, though they had never been fed, 13 lived 14 days, and one 20 days. Of 75 fleas which had sucked blood, 19 were alive at the end of 10 days; but they were all dead at the end of 16 days except 4, one of which lived 21 days.

Though the rat-flea can leap a distance of 18 cm., it rarely travels far; one metre a day is about the rate of progress.

The Java rat-flea attacks man sometimes after 24 hours' starvation, but generally not until three days after being deprived of food.

Ants immediately prey upon fleas and their larvae, when they are confined together. Some fleas bred in captivity become infested with an acarus which fixes itself to the abdomen and thorax of the flea. This insect is probably the "hypopus stadium" of the Tyroglyphinae (cheese mite). Fleas harbouring these acari fed on the blood of a plague animal did not convey plague.

Iodoform does not kill fleas, nor prevent their bites; fleas starved for two days avoided for two hours skin the surface of which had been covered with cajeput oil, but this effect passed

off in five hours. Exposure of fleas for one hour to the vapour of carbon disulphide was necessary to ensure their destruction. Carbon monoxide required the same time. 60 out of 100 fleas remained alive after 4 hours' exposure to carbon tetrachloride vapour. Sulphur dioxide fumigation destroys fleas in half an hour. Steam at 60° C. kills them in that period.

*Transmission.*—A large series of experiments was carried out to ascertain how long rat-fleas remain infective after feeding on a plague rat; 28 days was the limit. Fleas kept without food were infective till the sixth day. Three infective fleas were sufficient to excite the disease in rats.

Seven series of tests were made, in which 20-40 infected *X. cheopis* were placed on healthy rats and guinea-pigs, 14 to 42 animals being used in each experiment; 93-100 per cent. of the guinea-pigs died of plague, and 55-74 per cent. of the rats.

The *B. pestis* was found in the midgut of *X. cheopis* until the 18th day after its infecting meal, and in the rectum till the 15th day. But fleas may transmit plague by their proboscis as well as by their dejecta; for 25 infected fleas were induced to bite the shaven skin of three guinea-pigs through a layer of gauze: all the animals died of plague although no faecal matter came in contact with them.

The flea, *Pygiopsylla ahalae*, is found in much smaller numbers than the *X. cheopis*, and disappears entirely in the rains in some places. It can convey plague from rat to rat, but it does not bite guinea-pigs. Four out of 5 rats died of plague, after being bitten through gauze by *P. ahalae*.

Nine experiments were made in which from 7-254 body-lice removed from the clothing of plague patients were inoculated cutaneously into guinea-pigs; they all died of plague in 4-11 days, with the exception of the animal which received the 7 pediculi. *Cimex rotundatus* did not transmit the disease.

In several of these various transmission tests, more especially those with *P. ahalae*, the animal did not die for 3 or 4 weeks, when the appearances of chronic plague were found post mortem. A similar condition was induced in rats by inoculating them with a very small dose of infective material.

The *Trypanosoma lewisi* is conveyed by fleas; hence it would seem probable that the greater the number of fleas per rat the greater percentage of rats would be trypanosome carriers: curves are given showing that this is the case. But if there has been an epizootic of plague among the rats of a locality, we might expect a concentration of the fleas on the surviving rats, hence the flea curve would rise, while the trypanosome curve would remain stationary. Charts of plague areas where this has been observed are supplied.

The effect of human intercourse on the spread of plague was measured by counting the parasites in the clothing and baggage of 64,431 people who had been in contact with plague: 9,557 pediculi, 779 *Cimex rotundatus*, 237 *Pulex irritans*, 9 *Xenopsylla cheopis*, 9 *Ctenocephalus canis*, and 1 *P. ahalae* were captured.

*Summary.*—In Malang the rat-flea is the transmitter of plague; it remains infectious longer than in India, 28 days against 15 days. In East Java rat immunity is low. At Soerabaya it is greater.

The variations in the seasons are too small to exert an appreciable influence on the average number of fleas per rat, hence plague is not limited to one period of the year; sometimes it prevails in the rainy season and sometimes in the dry.

The spread of plague probably occurs through the carriage of house-rats in ships, boats, railway vans, and in merchandise generally, and also by their own migrations from house to house, and village to village. The *M. decumanus* and field-rat play little part in the diffusion of plague in Java.

It is possible that human lice may disseminate the disease.

Rat-plague may exist in a locality for months without man becoming infected; should therefore, human plague arise at any time, it does not follow that it has been imported from without.

There appears to be a connection between a high flea-count and rat-plague. A rising flea curve with a stationary trypanosome curve is suggestive of a rat epizootic.

For the prevention of plague, inspection of goods and control of their transport are essential; the control of human intercommunication is not so important.

C. B.

BROWN (B. W.). Plague. A Note on the History of the Disease in Hongkong.—*U.S. Public Health Rep.* 1913. Mar. 21. Vol. 28. No. 12. pp. 551-557.

Somewhat nebulous references to plague in China appear from the year 1844 onwards. The first epidemic in Hongkong began in May, 1894; it was introduced from Canton where plague was inflicting great ravages on the population. Some thousands of deaths occurred in Hongkong. In 1895 there were only 45 cases of plague among the population of about a quarter of a million persons. In 1896 there were 1,204 cases; in 1897, 21; in 1898, 1,320; in 1899, 1,486; in 1900, 1,086. In 1901 the campaign against rats was begun; 77,301 were caught. In 1902 there were 572 cases of plague; the whole of the sanitary staff, including 200 coolies, was inoculated with Haffkine's vaccine; no plague attacked them, though in the previous year 7 rat-catchers out of 30 died of the infection. In 1903 there were more than 1,300 cases; in 1904 there were 510. From 1905 to 1911 with the exception of 1908, the epidemics were small. During this period many houses had been rendered rat-proof, and rat destruction had been carried on, yet a severe outbreak began in January, 1912. From January to September there were 1,848 cases.

In all the epidemics the outbreak has usually begun in April, has reached its maximum in June, and has declined rapidly in July and August. This represents the rainy season, the maximum rainfall being in June. The large majority of cases has been of the bubonic type, the septicæmic form comes next, and pneumonic plague last in the order of frequency. The European has generally escaped.

Hongkong is infested with mice, *M. rattus* and *M. decumanus*.

The author concludes that bubonic plague is endemic, and rat infection is always present. Every vessel that is dry-docked, or goes to a wharf without proper precautions is liable to be infested with plague rats, and he states that the most important quarantine measure applicable to Hongkong is frequent sulphur fumigation of vessels.

C. B.

## PROPHYLAXIS.

FOY (F. A.). **Port Health Inspection as a Factor in Plague Prevention.**—*Jl. of State Medicine*. 1913. May. Vol. 21. No. 5. pp. 282-289.

In the years 1901-1908, 2,224,260 immigrants to Burmah landed at Rangoon; 23 cases of plague were detected among them, or 1 in 96,707. Since the abolition of the inspection of immigrants in 1908, 7 cases of plague are known to have occurred among 1,094,324 arrivals in the following three years, or 1 in 156,000. These included many immigrants from plague-free ports, but among 383,839 from infected ports 19 cases of plague were discovered in 5 years, or 1 in 20,202.

One in 56,000 of 1,299,187 people leaving Rangoon were plague-infected and 1 in 44,063 of 132,187 emigrants from Rangoon to ports out of India developed plague during the last three years.

The author therefore questions whether it is worth while to inspect passengers who are proceeding from an infected port, particularly when the duration of the voyage covers the incubation period of 5-7 days. The inspection of immigrants is necessary, if they arrive from plague areas.

C. B.

SIMPSON (Friench). **Bat Proofing. Its Practical Application in the Construction or Repair of Dwellings or other Buildings.**—*U.S. Public Health Rep.* 1913. Apr. 11. Vol. 28. No. 15. pp. 679-687. With 20 figs.

Walls should be at least 6 inches thick when made of brick or concrete, and should extend not less than 18 inches below the surface of the soil. Floors should be composed of concrete 3 inches thick, with a layer of half an inch of cement on the surface. Ventilation openings must be screened with cast-iron grating, or wire gauze not less than 20 gauge, and not greater than  $\frac{1}{2}$  inch mesh. All openings round wires and pipes must be sealed with cement.

C. B.

## TREATMENT.

CONNOR (F. Powell). **The Results obtained in a few Cases of Bubonic Plague by the Intravenous Injections of Dilute Solution of Iodine.**—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. Part 2. pp. 148-149.

Six intravenous injections of one drachm of a solution of iodine, which consisted of seven parts of tinct. iodi and sixty parts of distilled water, were given in the course of two days to a patient in whose blood plague bacilli were seen microscopically; although he was suffering from plague in its severest form, he recovered. Two boys also were treated in a similar way, but with somewhat smaller doses; they both made perfect recoveries.

C. B.

## RAT-FLEAS IN ENGLAND.

- i. NUTTALL (George H. F.). **Observations on British Rat-fleas July-October, 1911.**—*Parasitology*. 1913. Apr. Vol. 6. No. 1. p. 1.
- ii. STRICKLAND (C.) & MERRIMAN (G.). **Report on Rat-fleas in Suffolk and North Essex.**—*Ibid.* pp. 2-18. With 3 charts.
- iii. NUTTALL (George H. F.) & STRICKLAND (C.). **Report on Rat-fleas in Cambridgeshire.**—*Ibid.* pp. 18-19.

i. The investigations described in ii and iii were undertaken in Suffolk, North Essex, and Cambridgeshire. The fleas were collected in the field immediately after the rat had been caught and killed.

ii. In 1910 MARTIN and ROWLAND found that rat plague prevailed chiefly in those parts of Suffolk where fleas were most numerous on rats, and they isolated plague-like organisms from the intestine of fleas infesting plague rats.

Rats were put into calico bags immediately after being killed, and the bags were placed in tins, into which chloroform was dropped. The fleas abandoned their host and died in a few minutes; they were preserved in 50 per cent. alcohol. To render fleas transparent for microscopical examination, they are transferred from alcohol to a strong phenol solution, in which they are left a day; then after being treated with clove oil for a few minutes, they are mounted in balsam.

*M. decumanus* was the only rat caught, and it was scarce in the summer. 75 per cent. of 822 rats harboured fleas; on one there were 105, and on several 30 or 40 fleas. Two charts are given in which it is seen that the rat-fleas decrease as the temperature and humidity of the air decline. It has been proved that *C. fasciatus* can convey plague from rat to rat, and that it bites man, but the authors could find no correlation between the average number of *C. fasciatus* per rat and rat plague. It deserts dead rats later than other species; it is also a parasite on mice and rabbits. *C. agyrtes*, although it does not attack man, is found on mice, and may spread plague epizootics. The rabbit-flea, *Spilopsyllus cuniculi*, said to bite man, and *C. musculi*, the mouse flea, are caught sometimes on rats. Lice were observed on 5 rats only.

## Summary.—

(1) We found an average of four fleas per rat: 822 rats were examined and 3,293 fleas taken.

(2) The average was subject to a local variation (0.6 to 6.5).

(3) 15 species of fleas were taken from the rats, but of these there were only two species, *C. fasciatus* and *C. agyrtes*, which occurred in any numbers.

There were—

<i>C. fasciatus</i>	1,986,	or about	60 per cent.
<i>C. agyrtes</i>	1,257	„	38 „
Rare species	50	„	2 „

(4) The average number of fleas per rat and the percentage of rats infested by fleas showed a well-marked seasonal variation, there being a considerable decline in the numbers as the cooler weather came on. *Ceratophyllus fasciatus* and *Ctenophthalmus agyrtes* both participated in a similar way in this variation.



(5) *C. fasciatus* is chiefly found on rats caught near human habitations; *C. agyrtes* on those caught in the hedge-rows.

iii. 297 fleas were obtained from 150 rats caught in Cambridge and its neighbourhood; they were mostly *C. agyrtes*, which were found in largest numbers during February and March. A single specimen of *P. irritans* was secured.

C. B.

#### UNCLASSIFIED.

BARBER (M. A.). **The Susceptibility of Cockroaches to Plague Bacilli inoculated into the Body Cavity.**—*Philippine Jl. Science*. Sect. B.—*Philippine Jl. Trop. Med.* 1912. Dec. Vol. 7. No. 6. pp. 521-524.

Inoculations of about two and a half millions of virulent *B. pestis* were made into the legs of 61 cockroaches, *Periplaneta americana* and *Rhyparobia maderae*; six died of unmixed plague infection within two days.

The author finds that cockroaches may be infected by large doses of virulent plague bacilli, but from the fact that massive doses failed to infect a large proportion of cases it may be concluded that these insects, especially *Rhyparobia maderae*, are little susceptible to plague inoculated into the body cavity.

C. B.

HOSACK (D. C.). **Some Lessons of the Manchurian Plague Epidemic.**—*Jl. of State Medicine*. 1913. Apr. Vol. 21. No. 4. pp. 228-233.

The value of this article can be assessed by quoting the author's own words: "I maintain that the time has come when, even in England, it must be acknowledged that the rat-flea theory is an erroneous one that has failed to stand the test of practical experience."

There is no record of any experimental work by him in the transmission of plague.

C. B.

DE RAADT (O. L. E.). **De critiek van Dr. Kiewiet de Jonge op mijn oordeel over het Pestvaccin.**—*Geneesk. Tijdschr. v. Nederlandsch-Indie*. 1913. Vol. 53. No. 1. pp. 155-164.

DE JONGE (G. W. Kiewiet). **Antwoord aan Dr. de Raadt.**—*Ibid.* 1913. Vol. 53. No. 1. pp. 165-172.

After making somewhat arbitrary assumptions, de Raadt modifies the statistics of inoculation against plague which he gave originally (see this *Bulletin*, Vol. 1, p. 317). By so doing he has reduced the incidence of plague among the uninoculated from 0.86 per cent. to 0.68 per cent., and has increased the incidence in those who were inoculated with the Dutch vaccine from 0.26 to 0.5 per cent.; and in those inoculated with Haffkine's prophylactic from 0.16 to 0.31 per cent. He states that it has not

been proved that the Dutch vaccine confers the least immunity against plague.

After criticising his statistical methods, de Jonge traverses de Raadt's last statement. Of 1,688 not-inoculated who were seized with plague, 1,363 died; and of 133 who had been inoculated with the Dutch vaccine, subsequently attacked with plague, 105 died; that is, the mortality was 1·8 per cent. less in the inoculated than in the uninoculated. On working out the probability, he finds that the chances are over 2 to 1 that the slightly diminished mortality of the vaccinated plague cases is causal and not accidental.

[No detailed statistics are given, hence these polemical papers are not of much worth.]

C. B.

SAISAWA (K.). i. Ueber die Pseudotuberkulose beim Menschen. [Pseudo-tuberculosis in Man.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. Vol. 73. No. 3. pp. 353-400. With 4 plates.

ii. Vergleichende Untersuchungen über den Bacillus der Pseudo-tuberkulose. [Comparative Experiments with the *B. pseudo-tuberculosis*.]—*Ibid.* pp. 401-420.

i. ROWLAND pointed out the close relation which exists between the *B. pseudo-tuberculosis rodentium* of PFEIFFER and *B. pestis* (see this *Bulletin*, Vol. 1, p. 544). The author observed a case of fever in Japan in the year 1909, in which the symptoms were those of enteric fever complicated with tonsillitis and jaundice, which ended fatally on the eleventh day. The Peyer's patches were greatly swollen, and ulceration was beginning. Grey points, the size of a head of a needle, were visible in the interior of the liver and spleen. The *B. pseudo-tuberculosis* was isolated from the blood withdrawn on the tenth day of the fever, and from fluid aspirated from the pericardium on the day after death, but many contaminating bacteria were obtained from the other post-mortem material. The author nevertheless concludes that the organism which he isolated from the blood during life was the cause of death, and that it is identical with the *B. pseudo-tuberculosis* of PFEIFFER.

ii. The original *B. pseudo-tuberculosis rodentium* of PFEIFFER; a culture isolated from guinea-pigs by ABEL; a culture obtained by LOREY from the blood of a man who was suffering from an enteric-like fever and jaundice; a culture isolated by ALBRECHT from the ulcerated portion of the ileum of a man which had been resected during life; and the author's bacillus; were submitted to comparative bacteriological and biological tests. No marked differences were discovered between them. They were all strongly pathogenic to rabbits, guinea-pigs, and mice; but rats resisted subcutaneous inoculation. Vaccination with one strain protected the animal from an otherwise fatal dose of another.

C. B.

GALLI-VALERIO (B.). *Bacterium pseudopestis murium* n. sp.—  
*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Mar. 1. Vol.  
68. No. 2. pp. 188-194. With 5 figures.

A rat died three weeks after drinking spring water from the Jura, suspected of causing goitre; its spleen was enlarged and there was an abscess in its thyroid gland. From the pus a bacillus which closely resembled the *B. pestis* was isolated: the growths however were not so abundant as those of *B. pestis* and they soon died. Half a cc. of a broth culture inoculated subcutaneously caused the death of a *Mus rattus* in 70 days; in which the post mortem appearances were emaciation, loss of hair, enlarged spleen, and abscesses in the thyroid gland and lung from which the micro-organism was recovered and gave rise to similar lesions in other rats three or four months after inoculation; sometimes also the testes were enlarged.

Although cultures were not obtained directly from the spring water, the subcutaneous injection of half a cc. of the centrifuged deposit caused a fatal infection to a *M. rattus* after three months.

A guinea-pig was refractory to one cc. of a broth culture. A rabbit died two months after an injection of one and a half cc.; it became emaciated and lost most of its hair.

The bacillus probably belongs to the group *B. pseudo-tuberculosis rodentium*.

*Summary.*—

The name *B. pseudopestis murium* is proposed for an organism isolated from the lymphatic glands, thyroid, testes, and spleen of a *Mus rattus*.

The infection probably was conveyed in spring water from the Jura.

The thyroid gland is often invaded by the bacterium.

C B.

ERRATUM.

*Bulletin* Vol. 1, p. 316, line 8, for "feet [sic]" write "gut." Capt. Knowles writes that this was the word used in his MS.

## BERIBERI.

VEDDER (Edward B.) & CLARK (Elbert). **A Study of Polyneuritis Gallinarum. A Fifth Contribution to the Etiology of Beriberi.**—*Philippine Jl. of Science. Sec. B. [Philippine Jl. of Trop. Med.]* 1912. Oct. Vol. 7. No. 5. pp. 423-458. With 11 plates.

The authors describe the symptoms and pathology of polyneuritis gallinarum, illustrating the latter by 11 photo-micrographs which are excellently reproduced. In a summary of the work it is noted that three types of the disease are found: (1) A form in which the symptoms of neuritis and general prostration are combined; this is the usual form. When the birds are given an extract of rice polishings they improve at once in general condition; but the symptoms of neuritis only disappear after several months. (2) A form in which there is pronounced neuritis but the bird remains in good health. These recover after several months of treatment. (3) Fulminating cases, in which the symptoms of neuritis are absent, but in which greater general prostration occurs; these birds speedily recover when given extract of rice polishings.

After prolonged feeding on polished rice very marked degenerative changes are found in the vagus, sciatic, and peripheral nerves, affecting both the myelin sheath and axis cylinders. Degenerative changes are also observed in both dorsal and ventral roots, in the fibres of all the columns of the thoracic spinal cord, and in the large cells of both ventral and dorsal horns of the lumbo-sacral cord. When fowls, fed on polished rice, received a small amount of protective substance, all the evidence of nerve degeneration came on after a prolonged incubative period, from 43 days to one year of such feeding. Ten grams of white wheat, or 5 c.c. of canned milk added to the diet of polished rice gave little or no protection; a small quantity of meat, potatoes, or fresh cows' milk gave partial protection; but 10 grams of dried peas or peanuts gave complete protection for at least 60 days.

The following conclusions are given:—

1. In addition to the changes demonstrated above, FUNK has shown that chemical changes take place in the brains of fowls suffering from polyneuritis gallinarum. It therefore appears that the disease is not simply a peripheral neuritis as has been generally supposed. On the contrary, the entire nervous system is affected.

2. The symptoms of the disease are not chiefly referable to degeneration of the peripheral nerves, since the degeneration occurs before symptoms arise, and because advanced degeneration may be present accompanied by no symptoms at all, and because degeneration of the nerves remains after recovery has occurred.

The authors do not regard polyneuritis gallinarum as identical with beriberi in man, but experiments show that the cause is the same, viz. the want of the neuritis preventing substance in the food; "the two conditions are due to pathological processes causing slightly different manifestations in diverse species" (VEDDER and

CHAMBERLAIN). The authors deduce from the feeding experiments on fowls important facts concerning the relation between diet and beriberi in man. As meat and potatoes contain a small quantity of the neuritis preventing substance, those who subsist almost entirely on them are free from beriberi: while if these are only added in small quantities to a diet deficient in protective substance, the symptoms are manifested. Wheat bread and biscuit made from over-milled wheat flour are deficient; ship beriberi develops in those living mostly on the latter, and beriberi among the native population of Labrador who live during certain seasons almost exclusively on the former. It is also important to note that peas and peanuts possess the property of preventing the disease, and can be used prophylactically on board ship; the fact that cooked meat and potatoes afford more protection than when taken raw is at present unexplained.

The organic base, called vitamine by FUNK, is accepted as the essential substance required for normal metabolism of nervous tissue; if this is reduced even slightly below the requirements of the particular subject, degeneration of nerves occurs and if continued, neuritis follows. In cases where there is a great loss of weight without neuritis it is possible that this is due to a deficiency of fat, phosphorus, or potassium. The very great prostration sometimes found may be due to degeneration affecting the higher nerve centres in the brain or cord. It is suggested that there may be two vitamins which are essential for proper metabolism of the body, one the neuritis preventing substance, the other a substance which prevents general prostration, cardiac failure, &c.: this hypothesis would account for the three classes of symptoms found in fowls, and would explain the cases of ship beriberi without neuritis, and possibly the disease known as "epidemic dropsy."

[This paper should be read in the original by all interested in the subject.]

P. W. Bassett-Smith.

YAMAGIWA (R.), KOYANO (T.), MIDORIKAWA (H.), & MOGI (T.).

**Experimental Study of the Cause and Nature of the Beriberi.**

**Report 1.** (The original in No. 23, Vol. 26, 1912, of the *Jl. Tokyo Med. Assoc.*)—*Sei-I-Kwai Med. Jl.* 1913. Feb. 10. Vol. 32. No. 2. pp. 12-15.

The object of these experiments was to show whether it was possible to produce in fowls, by subcutaneous injections of artificially fermented solutions of polished rice, the polyneuritic symptoms commonly seen when these birds are fed on polished rice. The solution for injection was obtained by placing 2 litres of the rice with 800 c.c. of water in a shallow dish for 48 hours at 40°C. The following results are recorded. A proportion of the fowls so treated showed symptoms of the disease; in addition to the polyneuritis a certain amount of cyanosis of the comb and diarrhoea were produced. The unsterilised solution after fermentation for 48 hours was more powerful than after 24 hours; it was still efficacious after passage through a Chamberland filter and sterilisa-

tion. It was found that simple infusions of rice given subcutaneously did not produce the same symptoms, but a dilute solution of acetic acid of equal acidity to that of the fermenting rice solution when injected quickly caused the fowls to die without producing any trace of paralysis. The post mortem changes found in three fowls examined were dilatation of the heart, congestion of the internal organs, and degeneration of the nerves, muscles, and arteries, conditions similar to those found in fowls fed on polished rice; there was also a catarrhal state of the intestinal mucous membranes. From their experiments the authors consider that the symptoms and pathological changes in birds fed on polished rice and injected with fermenting rice solutions coincide, also that some birds are more refractory than others to both measures, the difference depending upon the affinity of the tissues for the poison. The following conclusions are given:—

(1) The disease produced by the injection of a fermented solution of polished rice is an intoxication.

(2) This intoxication disease and the beriberi-like disease of fowls caused by feeding are the same.

(3) As the former is absolutely proved to be an intoxication, so must the latter be.

(4) The beriberi-like disease of fowls caused by feeding with polished rice is not a so-called "partial inanition."

P. W. B.-S.

FUNK (Casimir). **An Attempt to estimate the Vitamine-Fraction in Milk.**—*Biochemical J.* 1913. March. Vol. 7. No. 2. pp. 211-213.

The author, in continuation of his experimental work on the food problem and its relation to polyneuritis in birds and beriberi in man, has in this paper drawn attention to three lines of research. (1) What is the normal amount of vitamins in milk of different species, including the human? (2) Is there a definite relationship between the amount of vitamins secreted in the milk and that ingested in the food? (3) What effects have boiling and pasteurisation on the vitamin content of milk?

The experiments here described show that ordinary chemical methods for estimating vitamins are not sufficient; colorimetric methods are therefore required. From 1 litre of milk 1.3 cgm. of vitamin were obtained, giving 1.25 mgm. of nitrogen; the residual nitrogen, amounting to 20-50 mgm., represents probably allantoin; therefore 1 litre of milk contains 0.06-0.15 grm. allantoin, which agrees with the amount found by ACKROYD (0.199 grm.). Artificial protein-free milk, as was used by OSBORNE and MENDEL (1912) in their experiments on growth, differs from true protein-free milk in being deficient in these nitrogenous substances, which may play an important part in process of growth. The experiments show that milk which has been deprived of its fats by centrifuging loses about 50 per cent. of vitamin and allantoin; also that allantoin, like vitamin, is destroyed by boiling.

P. W. B.-S.

FUNK (Casimir). **Studies on Beri-Beri. Further Facts concerning the Chemistry of the Vitamine-Fraction from Yeast.**—*Brit. Med. Jl.* 1913. April 19. p. 814. With 1 plate.

The author, in 1911, described the preparation of a vitamine fraction from rice polishings, which was effective both as a curative and a protective agent in birds suffering from polyneuritis caused by feeding on polished rice; a single substance was then isolated. In 1912 a similar vitamine fraction was obtained from milk, yeast, brain, and lime juice, but this was less active than the crude product. Since that time other purin and pyrimidin derivatives have been shown to be present, possessing slight curative effects. From 100 kg. of dried yeast the total amount obtained was 2.5 grams. A series of experiments is described, showing that birds badly paralysed, which would have died in less than 24 hours, were cured by injections of 4 to 8 mgm. of the vitamine fraction. If kept on the same diet they again developed the severe symptoms, showing that the injected substance was soon used up for metabolic processes; a fresh injection again effected a cure. By further experiment the author has been able to separate three distinct substances, which have been purified and analysed. The observations of FÜHNER on the composition of pituitary extract are somewhat similar, for he found four substances to be present, each having a specific action, but the effect of the original extract could only be obtained by the collective action of all four; it remains to be proved whether the same is the case in the vitamine fraction or whether one substance plays a preponderating part in the process of cure. For therapeutic purposes the best results at present may be expected from the use of the whole vitamine fraction, especially in acute cases.

P. W. B.-S.

TSUZUKI (J.). **Eine Beriberiepidemie auf Fischerbooten bei den Tsishima-Inseln (Japan).** [An Epidemic of Beriberi in the Fishing Fleet at the Tsishima Islands.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. May. Vol. 17. No. 9. pp. 306-308.

A description is given of an epidemic of beriberi which occurred in the fishing fleet working between Jesso and Kamtschatka. The disease broke out in August, and lasted throughout September and October. The men employed were all Japanese, mostly from the district of Awa, and when affected were removed to their homes for treatment. The author, Director of the Beriberi Institute at Tokio, was sent to investigate the cause of the epidemic, and to institute measures for the prevention of further outbreaks. There were altogether 249 men employed; 92 suffered from the disease, and 19 died. It was found that all the severe cases occurred in the vessels of the Enyo company, which supplied rice as the staple diet for their crews, whereas the other boats had a rice-barley diet: to the preponderance of rice diet on the ships of the Enyo company the epidemic was mainly due.

A table is given of the incidence and death rate in the different companies employed, which is highly interesting.

Name of Company.	Number of vessels.	Number of crews.	Number of cases.	Number of deaths.
Enyo ... ..	10	186	73	19
Niigata ... ..	2	47	16	0
Hoko ... ..	1	16	3	0
Total ... ..	13	249	92	19

P. W. B.-S.

GREGG (Donald). *Infantile Beriberi in the Philippines.*—*Boston Med. & Surg. J.* 1913. May 8. Vol. 168. No. 19. pp. 676-678.

The author gives an historical and clinical history of the disease, gathered mostly from the works of previous writers, more particularly those of CHAMBERLAIN, VEDDER, and ANDREWS; these have already been reported on in this *Bulletin*. From his own experience he emphasises the suddenness with which an apparently healthy baby may die from an acute cardiac attack. The prophylactic measures recommended are: (1) Sufficiently nourishing diet to the nursing mother. (2) Substitution of artificial feeding for breast milk. (3) The administration to the infant of extracts of rice polishings, locally called "tiqui-tiqui"; the preparation is sweet and pleasant to take, and efficacious and rapid in its action; 5 cc. are given daily. The last method is often the only one practicable.

P. W. B.-S.

GENUARDI (G.). *Alcuni Casi di Beri-beri osservati sulla Regia Nave "Calabria," con Considerazioni Cliniche ed Eziologiche.* [Some Cases of Beriberi in the Royal Naval Ship "Calabria."]—*Ann. di Med. Navale e Colon.* 1913. Jan. Anno 19. Vol. 1. No. 1. pp. 12-22.

Seven cases of beriberi are described from a clinical standpoint, and some of the etiological factors are noted. All the cases occurred on board the R.N. ship "Calabria," which had been for two years in the far east, had visited China and Japan, and proceeded to Massawa (Red Sea) in February 1912. Details are given; the cases were mostly of the oedematous type and none were fatal. The most interesting points noted are the following: the disease did not appear until 4 months after leaving China, or 5 months after leaving Japan, and other cases occurred in the "Puglia" and "Volturne," also in the Red Sea, these ships not having been to the far east but being in communication with the "Calabria." With regard to the food, it is stated that the nutrition value of the rations served out was unknown; rice was provided four times a week, but it did not constitute the principal food. The predisposing causes were:— (1) Great heat



and humidity, which may favour the growth of a germ. (2) Excessive fatigue in a tropical climate. The conditions under which the men lived at Assab and in the Red Sea were said to have been very trying. On the appearance of the first case the issue of rice was stopped, and the epidemic quickly ceased.

P. W. B.-S.

**MONTEFUSCO (A.).** *Su Due Casi di Beri-beri. Osservazioni Cliniche.* [Clinical Observations on Two Cases of Beriberi.]—*Ann. di Med. Navale e Colon.* 1913. Jan. Anno 19. Vol. 1. No. 1. pp. 23-30.

The author gives a short summary of the geographical distribution of beriberi and its occurrence in the Italian Navy. He describes two cases, admitted from the "Piemonte" and treated in the hospital at Massawa. The results of the blood examinations are given: in one there was a marked eosinophilia, in both an increase in the mononuclear leucocytes. He states that the cases furnished no new clinical or etiological facts, but as rice is very little used in the Naval ships at Massawa, it was probably not the causative agent.

P. W. B.-S.

**RICHER.** *Rapport sur une Epidémie de Béribéri a la Prison de Loango (1911-1912).*—*Ann. d'Hyg. et Méd. Coloniales.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 145-156.

The occurrence of several small epidemics of beriberi in the prison at Loango, Congo Française, forms the subject of this paper. During March, 1911, four cases were recognised; these were of the wet form, and all recovered. Steps were at once taken to stamp out the disease: the existing diet, of white rice and dried fish, was improved by the addition of fresh meat, the prison was disinfected, and the prisoners were made to exercise in the open air. No fresh cases occurred until the end of August, when a number of Bayakas were received; these prisoners were in a miserable condition, and 7 days after their admission eight developed marked gastric symptoms, eight definite cases of beriberi were treated in the hospital, and three died. After considerable difficulty fresh provisions were supplied, and the disease did not increase. The supplies of fresh food (manioc) then failed, and beriberi again broke out in January (11 cases, with 2 deaths). Fresh manioc, fruit &c. was once more procured, and the epidemic ceased. Clinically the forms were associated with marked cardiac symptoms and much oedema; phlebitis of one of the deep veins of the thigh occurred in one chronic case, this being followed by suppuration, and death of the patient. A post-mortem was made in one case only, but nothing definite was determined. The author states that the difficulty of providing fresh provisions in the prison was very great, but that there could be little doubt that the outbreaks of the disease were due to the deficiency of fresh food, and were favoured by the debilitated condition of many of the prisoners.

P. W. B.-S.

COZANET. **Relation d'une Epidémie de Béribéri ayant sévi à Gao (Haut-Sénégal-Niger).** — *Ann. d'Hyg. et Méd. Coloniales.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 157-169.

The author, who was sent to investigate an epidemic of beriberi which had broken out at Gao, gives a detailed account of the cases which occurred, and the measures employed to stamp it out. Thirty cases were noted in the tirailleurs, with four deaths: clinically they were all of the moist form, and were not very severe. The measures used were, substitution of millet for rice, thorough disinfection of the quarters, isolation of all cases, and inspection to detect early forms. The author's view was that the epidemic was not due to alimentary intoxication, but to an infective agent not yet known.

P. W. B.-S.

DOYLE (Stanley B.). **Beriberi. A South American Aspect of the Disease.**—*New York Med. Jl.* 1913. Apr. 19. Vol. 97. No. 16. pp. 828-829.

This disease, the author says, is extremely prevalent along the west [east] coast of South America, from the Amazon river in the north to Rio del Plata in the south. A short account of the disease, as usually met with, is given and two cases are described in detail.

P. W. B.-S.

SCHUFFNER (W.). **Ist die Beriberi eine auch in Europa heimische Krankheit?** [Is Beriberi endemic in Europe?]*—Munchen. Med. Wochenschr.* 1913. Mar. Vol. 60. No. 12. pp. 642-647.

The author, from his experience in the Dutch East Indies (Deli), shows that beriberi has ceased to be epidemic there, and now causes practically no mortality. He describes the symptoms of the disease and goes fairly fully into the food deficiency theory, which he practically accepts, but he asks—Do the protective substances act as (1) food stuffs, (2) as building stones in the molecular synthesis, or (3) have they a specific effect on internal secretions, playing the part of hormones? He believes that beriberi is not epidemic in Europe owing to the fact that the food used is never absolutely deficient in the protective substances; such articles as bread and potatoes are fairly rich in proteids and salts, and therefore probably contain the essential bodies; the disease however may occur sporadically from errors of diet and destruction of the protective substances by bacterial action. It is probable that the polyneuritis following typhoid fever is due to the restricted diet, the pseudotabes of diabetes to the cutting off of the hydrocarbons, the neuritic condition of sucklings to the imperfect nourishment of the pregnant woman—these conditions being forms of secondary beriberi. A good many of the alimentary and developmental affections incident to early childhood may very well be initial stages of beriberi. The author thinks that all cases of progressive polyneuritis found in Europe should be carefully examined with

regard to their etiology and treatment. MAURER, OPPENHEIM, and DÜRCK have each suggested the possibility of beriberi occurring in Europe.

[This paper is mostly theoretical and contains no new facts.]

P. W. B.-S.

STRONG (Richard P.) & CROWELL (P. C.). **The Etiology of Beriberi.**—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 195-198.

This is a resumé of the experiments carried out on a number of prisoners under sentence of death, to prove whether beriberi in man is an infectious disease, or, like the polyneuritis gallinarum (EDLMAN), is due to a deficiency in the food. The conclusions arrived at were definitely in favour of the latter, and confirmed the early work of FRASER and STANTON. The original paper in the Philippine Journal of Science (Vol. 7, No. 4, 1912) has been already reported upon in this *Bulletin* (Vol. 1, p. 477).

P. W. B.-S.

LE DANTEC (A.). i. **Note sur un Mycoderme rencontré dans les Fèces de deux Matelots Béribériques.**—*Compt. Rend. Soc. Biol.* 1913. Feb. 28. Vol. 74. No. 8. pp. 412-413.

ii. **Mycodermose Intestinale dans divers États Pathologiques.**—*Ibid.* pp. 414-415.

(i) In December 1911, during the examination of the faeces of two Europeans suffering from ship beriberi, the author recognised the presence of a mould, which has not been found before in the intestinal flora of man. The two cases came from the "Antoinette," on board which ship there was said to be an epidemic of beriberi; the men were treated in the hospital at Nantes. A full description of the cultural characters is given, and it is stated that the stab culture in gelatine is very similar to that of *B. anthracis*, showing lateral outgrowths, and causing very slow liquefaction of the medium. Growths on glucose agar had a grey colour, were thick and moist, and had a wavy outline. Aerial hyphae were not found. Milk became slowly acid, and gave off a foetid odour. The organism injected under the skin is not pathogenic to rats, guinea-pigs, and fowls; intra-peritoneal injections into guinea-pigs produced a peritonitis which was not fatal.

The author, in conclusion, states that the presence of mycodermis in the intestine of beriberi cases has no specific value; it indicates only that the intestinal secretions are unable to prevent their growth if they are introduced in food.

(ii) The above-mentioned discovery led the author to examine for the presence of the same micro-organism in other pathological states. Similar organisms having no specific differences were found abundantly in the faeces of six mental cases, namely, three melancholics, one epileptic, and two others.

P. W. B.-S.

OGATA (C.). **On the Blood Vessels by Beriberi.** (To be followed.)  
[The original in Nos. 13, 14, Vol. 26, of *Jl. Tokyo Med. Assoc.*—*Sei-I-Kwai Med. Jl.* 1912. Nov. 10. Vol. 31. No. 11. pp. 245-247.]

From a study of the blood vessels of beriberi and non-beriberi cases the author came to the following conclusions.

1. Rigor mortis of the vessels appears 2 to 3 hours after death and reaches its maximum in 7 to 15 hours; it then gradually disappears and after 24 hours the vessels are absolutely relaxed. In beriberi the contraction is more marked than in non-beriberi cases. 2. There is some irregularity in the tunica media of the vessels in beriberi, with spaces between the cells. 3. In one case of beriberi a transmigration of the cells of the involuntary muscle fibres was recognised in the dorsal artery of the foot. 4. There was no rupture of the elastic fibres as mentioned by Glogner. 5. In beriberi there was a slight thickening and fatty degeneration of the tunica intima of the large arteries, and a swelling and vacuolation of the endothelial cells was observed. 6. Thrombosis of the femoral artery was present in 2 out of 8 cases of beriberi, and marked tortuosity of the pulmonary vessels was found in 3 out of 8 cases. 7. The follicles of the spleen showed a hyaline degeneration in all cases of beriberi.

P. W. B.-S.

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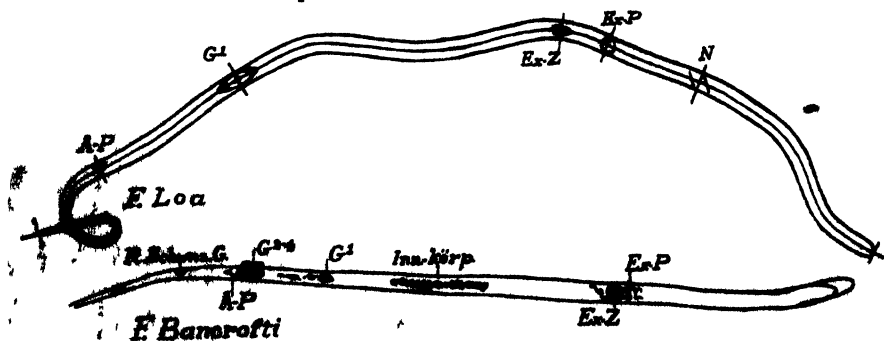
## FILARIASIS.

FÜLLEBORN (R.). Beiträge zur Morphologie und Differentialdiagnose der Mikrofilarien. [Morphology and Differential Diagnosis of Microfilariae.]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Jan. Vol. 17. Beiheft 1. 72 pp. With 8 plates.

This very important paper brings our knowledge of the morphology and differential diagnosis of the human microfilariae to date and collects all that is known about the subject within a reasonable compass. As regards fixing methods, that of Looss, which consists of using 70 per cent. alcohol heated to 50 or 60° C., may be added to the older ones and the author's suggestion of quickly dried thin films is also a useful one.

Staining may be accomplished by (a) Vital staining with neutral red, (b) Staining with Azur II or (c) other methods, haematoxylin, etc.

After the finer anatomy of the head and cuticle has been dealt with, Fülleborn passes to the excretory organs. These are the *Exkretionporus* [excretory pore or V spot of the English]; the *Exkretionszelle* [excretion cell]; the *Analporus* [anal pore, genital pore or tail spot] and the *G. zellen* [G. cells or genital cells of RODENWALDT]. The importance of these as landmarks for measurements will be detailed later. The tail part of the worm, the position of the cells in which is of importance, is mentioned in detail, as are also the length and thickness of the microfilariae, these being of course differential diagnostic characters of great value. The relative lengths of the anatomical fixed points in the different species are also very useful for differential diagnosis. Fülleborn proposes a scheme of measurement which seems to be a sound one and should be universally adopted. He takes the following points for making measurements from:—1. The middle of the nerve ring (N.) [this corresponds to the break in the continuity of the cells of the English]; 2. The middle of the excretion pore (Ex-P.); 3. The nucleolus of the excretion cell (Ex-Z.); 4. The nucleolus of the G<sup>1</sup> cell (G<sup>1</sup>); 5. The middle of the anal pore (A-P.); 6. The termination of the last tail cells, haematoxylin staining. In addition the length of the hinder end of the red tail structure (*das rote Schwanzgebilde*) and the commencement and ending of the stained inner body (*Innenkörper* or central viscus) may be noted. The following diagrammatic sketches make these points clear:—



A table giving a comparison of the measurements of these points from the head, and also the distances between each point, in filarial embryos from the Island of St. Thomé, New Guinea, German East Africa, the Philippine Islands, and Samoa is appended. A comparison of some of these results would seem to show that the embryos from Samoa, the Philippine Islands, the Bismarck Archipelago and German New Guinea are all examples of *bancrofti*. Another table deals with the embryos of *Filaria loa* extracted from the uterus of the parent worm and embryos found in the peripheral blood by day, the so-called *Filaria diurna*. The differential diagnosis between the embryos of *bancrofti* and *loa* are next described. Some of these have already been noticed [see this *Bulletin* Vol. 1, p. 87 and pp. 417-419]. In addition to these, however, Fülleborn gives the following diagnostic points: -1. The G<sup>1</sup> cell of *loa* is recognisable by its large size, while in *bancrofti* it is much smaller; 2. By azur staining it is seen that the nucleolus of the excretion cell lies relatively far from the excretion pore in *loa* as compared with *bancrofti*; 3. In fresh living material, staining with neutral red brings out the Innenkörper or central viscus only in *bancrofti*, not in *loa*; 4. The percentage reckonings of the position of the anatomical fixed points give characteristic differences for the two embryos. For practical purposes Fülleborn thinks the following are sufficient for making a correct diagnosis.

—	<i>F. bancrofti.</i>	<i>F. loa.</i>
1. In thick, not too slowly dried haematoxylin preparations.	(a) Close spiral coils ( <i>Windungen zusammengerollt</i> ).	(a) Crumpled, in wavy lines ( <i>zerknittert</i> ).
2. In Azur II preparations	(a) G <sup>1</sup> cell relatively small.	(a) G <sup>1</sup> cell very large.

The differential diagnosis between the embryos of *Filaria perstans* and *Filaria demarquayi* may be made by the appearance of the tail. In the former the tail is blunt and the cells come up to the extreme end, while in the latter the tail is sharp and the extreme tip is free from cells.

A full bibliography is given. Further tables deal with the measurements of the anatomical fixed points in microfilariae from different parts of the world, and there are eight plates (some coloured) of drawings of the filariae and their anatomical features mentioned in the paper.

C. G. Low.

MAROTTE & MORVAN. *La Filariose au Bataillon de Sénégalais d'Algérie.*—*Arch. de Méd. et Pharm. Militaires.* 1913. Jan. Vol. 61. No. 1. pp. 1-66.

In a very long paper the authors give the results of their examination of a battalion of Senegalese troops in Algiers for filariae. Out of a total strength of 1,339 blacks (1,325, if 14

infants born in Algiers are excluded) 313 or 23·37 per cent. were found to be suffering from filariasis. In their work the authors had a certain amount of assistance from CAZANOVE, [see his paper on the same subject in this *Bulletin* Vol. 1, p. 82]. The three common species of filaria (*Filaria perstans*, *F. bancrofti* and *F. loa*) were encountered as the following table shows:—

<i>Filaria perstans</i>	in	67	per cent.
„ <i>bancrofti</i>	„	29	„ „
„ <i>loa</i>	„	0·9	„ „
Mixed infections	„	4·1	„ „

Another table\* gives the percentages of males and females infected, and the geographical areas from which the cases were recruited:—

		Sudan.	Upper Senegal and Niger.	Senegal.	Guinea.	Casamance.	Congo.	Ivory Coast.	Mauretania.	Various.	Total.
Men	Numbers ...	535	93	85	46	14	10	9	7	12	811
	Number with filariae.	169	22	25	16	13	7	3	6	2	263
	Per cent. ...	31	23	30	34	92	70	33	85	16	32·5
Women and children	Numbers ...	280	36	150	15	4	11	1	9	22	528
	Number with filariae.	29	6	5	1	2	1	0	3	3	50
	Per cent. ...	10·5	16·6	3·3	6·6	50	9	0	33	13·6	10·2

Different clinical manifestations due to *Filaria bancrofti* were noticed among the troops and the authors believe that *F. perstans* may also give rise to various troubles. A large portion of the paper is devoted to the manner in which the filaria is inoculated into man. The authors still believe, apparently, that the parasite may gain access by the ingestion of water, but do not bring forward any facts in support of this. The prophylaxis is also dealt with and a fairly complete bibliographical index completes the work.

G. C. L.

MEINHOF (Heinrich). *Zur Klinik und Morphologie der Filaria und Mikrofilaria loa (diurna)*. [Clinical and Morphological Study of *Filaria* and *Microfilaria loa (diurna)*.]—*Beihefte z. Arch. f. Schiffs- u. Trop-Hyg.* 1913. Feb. Vol. 17. Beiheft 2. 58 pp. With 8 text-figures.

A very interesting clinical description of a case of *Filaria loa* infection is given. The description is specially instructive, because it covers a period of many years and so brings out features that otherwise might have been missed. The patient, a lady, born in Germany in 1878, went to the Cameroons as the wife

\* Some of these percentages are obviously misleading, owing to the smallness of the figures

of a missionary in 1903. Four weeks after arrival she suffered from fever, probably malarial, and then again severely in 1905. After this attack multiple swellings, having all the characters of Calabar swellings, appeared. For the next years these swellings kept coming, headache was much complained of, and febrile attacks were frequent. In 1908 the heart, previously normal, became irregular. In May 1911 a male *Filaria loa* measuring 2 cm. in length was extracted from the left eye, *i.e.* six years after the first Calabar swellings were noticed. After this from time to time rigors with severe headache were complained of. In October 1911, the author made an examination of the patient in Tübingen, Germany, when he found a permanent acceleration of the heart's action with a slight enlargement of the organ to the left. No malarial parasites or filarial embryos were found in the blood; the eosinophiles were eight per cent. On the 15th of December a filaria about 1 cm. long appeared below the skin of the left upper eyelid but escaped before it could be removed. The blood at this date again showed no malarial parasites nor filarial embryos. On the 4th of January 1912, for the first time, *loa* embryos were found in the blood. A differential count of the leucocytes gave the eosinophiles as 22 per cent. On the 2nd of February 1912, a male adult two and a half cm. in length was removed from beneath the left conjunctiva of the left eye and on other days embryos were again found in the peripheral blood. Another worm, a female, was extracted in July 1912 and when the patient was last seen on the 25th of October 1912 swellings were still appearing.

If the date of the infection is taken as 1905, when the first Calabar swellings were noted, the first adult filaria appeared six years afterwards and embryos eight months after that, *i.e.* almost seven years from the beginning of the infection.

The second part of the paper deals with the morphology of the embryos and the anatomy of the male and female adults. Tables of measurements of the latter are given. The anatomy of the embryos and their points of distinction from those of *Filaria bancrofti* have already been fully dealt with in this *Bulletin* [see Vol. 1, pp. 417-419 and paper by FÜLLEBORN above].

Some measurements are next given of the embryos of *F. perstans*, which were also found in the blood of the case and a very good and full account of the literature of the subject concludes the paper.

G. C. L.

Low (G. C.). *Filaria loa* Cases: Continuation Reports.—*Jl. Trop. Med. & Hyg.* 1913. Apr. 15. Vol. 16. No. 8. pp. 118-120.

The clinical histories of three of the five cases of *Filaria loa* infection reported upon in January 1911 and February 1912\* are continued. They reveal the following interesting features:

Case III. (1911). Having spent the intervening year in a non-endemic part of Africa, this patient reported himself again in

\* *Jl. Trop. Med. & Hyg.*, Vol. 14, p. 5 & Vol. 15, p. 38.



London on April 7th, 1913. During the interval sensations attributable to a filaria had been noted once near the left eye and once subcutaneously in front of the left ear. The blood showed no embryos, but the eosinophilia recorded in 1912 still persisted and amounted to 13·2 per cent.

Case IV. (1911). From February 1911 until June 1912 the patient had resided in the endemic area of Northern Nigeria in which he had originally acquired his infection. During this tour Calabar swellings developed five times on the back of the right wrist. The blood, examined on 3rd September, 1912, showed an entire freedom from embryos as on the previous occasion, but the eosinophilia had now increased to 17 per cent.

Case V. (1911) had shown no clinical features when reported upon previously, Low having accidentally found a number of embryos in the blood. When re-examined on 8th April, 1913, the patient complained of a little breathlessness and there was a suspicion of precordial pain with slight palpitation. The blood now showed an average of 39 as compared with 36 embryos per 20 cmm. The patient left the endemic area five and a half years ago.

Other points of interest emphasised by Low from his study of these cases are:—

- (a) Infection apparently does not induce anaemia.
- (b) The total leucocyte counts are not influenced.
- (c) The eosinophile cells are increased absolutely as well as differentially and the eosinophilia is much more distinctly a high one than in cases of *F. bancrofti*.

R. T. Leiper.

#### CHYLURIA.

CORYLLOS (Pol) & PORTOCALIS (A.). Un Cas de Chylurie à Rechutes, très Probablement d'Origine Filarienne, sans Microfilaires ni Eosinophilie Sanguine. Examen Cystoscopique et Cathétérisme des Urèteres.—*Bull. et Mém. Soc. Anat. de Paris*. 1912. June. (58th year. 6th series.) Vol. 14. No. 6. pp. 264-273.

The patient had resided for many years in Egypt. At the age of twenty he had a chancre which was treated as a soft one; no secondary manifestations of syphilis appeared. In 1902, after a slight shivering fit, his urine became white like milk. A physician diagnosed chyluria and advised a sea voyage. Four days after embarking the chyluria disappeared and did not reappear again until 1909. In 1908 a sister of the patient also suffered from the same complaint. In 1909 a second attack, longer than the first, appeared. It was not preceded by any premonitory symptoms and the general health remained good during the whole duration of the crisis. Retention of urine owing to the coagulation of the urine in the bladder proved troublesome, however, and the viscus had to be washed out to evacuate the clots. In February 1912 a third relapse occurred and this one was specially studied by the authors.

The condition of the urine was fairly typical as regards colour and other characteristics but BRUMPT who examined it chemically

and bacteriologically failed to find microfilariae. [This does not of course negative a filarial origin, as embryos are as frequently absent as present in such cases.] A further examination of the blood by the authors themselves showed no filarial embryos (day and night examinations) and a differential leucocyte count gave very scanty eosinophiles. Wassermann's reaction was negative. In view of the fact that the patient had had some ulceration of his legs some years previously and that after that a feebly positive Wassermann's reaction had been obtained in Hamburg, an intravenous injection of salvarsan was given. After this the urine became almost clear, but possibly the rest in bed was responsible for the amelioration of the symptoms quite as much as the drug.

Cystoscopy and catheterisation of the ureters was practised. The former revealed a normal mucous membrane as regards colour, but here and there, at the level of the trigone and on the anterior wall, were what appeared to be dilated lymphatics, sinuous vessels of irregular calibre, of a white colour, similar to those so often seen in the bladders of pregnant females. Catheterisation of both the ureters only gave clear urine, but as the urine from the bladder at this time also came clear it was not possible to say where the chyle was actually coming from, though the authors believe it was from the dilated lymphatic vessels in the bladder and not from the kidneys. LÜDKE (*Munch. Med. Wochenschr.* 1908 No. 26) has reported a similar case where the urine from both kidneys was normal and the chyle undoubtedly came from the bladder.

The authors then discuss their case as follows:—

(1) Was it a true chyluria? (2) What was the origin and cause of the chyluria? Finally they conclude that it was of filarial origin with disappearance of the adult filariae and consequent absence of microfilariae from the blood and urine and of eosinophilia in the blood.

[This explanation is in all probability the correct one. The death of the adults in filariasis is not uncommon and then the results of their previous presence only remain. In such a case embryos are of course absent from the urine and blood and no reason for an eosinophilia continuing exists. The fact of the patient's coming from Egypt is strongly in favour of *F. bancrofti* being the cause of the condition.]

G. C. L.

LAFORGUE. *La Chylurie.*—*Jl. des Praticiens.* 1913. Mar. 1. Vol. 27. No. 9. pp. 129-130.

A typical case of chyluria is described. The patient, a native of Martinique, had had several attacks, the last ones being complicated with haematuria [haematochyluria]. Swelling of both testicles was present and a hydrocele on the right side. The method of JOUSSER, for the examination of chylous ascites, was applied to the urine with the following results. The urine cleared completely with Adam's liquid and with glycerine, partially with boiling alcohol and with alcohol and ether. The

suspended materials were completely insoluble in ether, chloroform, xylol and acetone. Heated with potash, almost complete clearing took place, the subsequent addition of ether causing the remaining suspension to disappear. These reactions, especially the one obtained with Adam's liquid, enabled the author to conclude that fatty matter—chiefly a neutral fat—was present in the urine. The total insolubility in xylol excluded lecithin. In addition albumin, globulin, and blood were present and microscopically a small number of cellular elements.

A rigid diet caused the disappearance of the chyluria, which reappeared again on the resumption of ordinary food. Fatty foods, such as lard and pork fat, always aggravated the condition and increased the output of fat. Alcohol also produced it, as did the addition of 150 grams of butter to an ordinary milk regime, which when taken alone was harmless (*régime achylurique*).

The author discusses the origin of the condition, but seems to be a little doubtful as to the *Filaria bancrofti* being the cause owing to the absence of embryos from the blood and urine. [This is quite common in such cases and is usually due to the death of the parent or adult worms. From the description of the case its filarial origin is clear, as is also the fact that the chyluria was a true one and not a lymphuria, the blockage being high up, probably in the thoracic duct itself.]

G. C. L.

#### FILARIAL ABSCESS.

KÜLZ (L.). Der tropische Muskelabszess (*Myositis purulenta tropica*). [Tropical Muscle Abscess.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1912. May. Vol. 16. No. 10. pp. 313-324.

Intramuscular abscesses are very common in the Cameroons. In the author's list of operations at the hospital at Duala for 1907-8, out of 386 natives eighteen were operated upon for muscle abscesses and out of 86 Europeans five. In the years 1909-10, 4 Europeans and 11 natives were treated for this condition, and in Kribi (South Cameroons) in nine months 3 white people and 5 natives. The abscesses were usually of moderate size, about the size of a hen's egg, and from some pure cultures of *Staphylococcus pyogenes albus* were grown. Sometimes they were multiple, or in other instances new ones kept developing for weeks, or even months. In most of the Europeans treated the abscesses were single, whilst in the natives they were generally multiple. The youngest patient seen was fourteen years old. The differential diagnosis had to be made between muscular rheumatism, rheumatic swellings and Calabar swellings due to the *Filaria loa*.

As regards treatment, when pus forms it should be evacuated. After this the temperature falls to normal and the abscess soon heals. No fatal cases were met with in Europeans but two natives died: one of these also suffered from severe leprosy, the other from dysentery. The author believes that these cases of purulent myositis are really due to filarial disease. He suggests that *Filaria loa* is responsible. [It is much more likely that the condition is caused by *Filaria bancrofti*. In the reviewer's experience of *Filaria loa* infections in Europeans abscesses do not occur.

Again the fact of similar abscesses being found in the South Sea Islands and in other areas (British Guiana, West Indies, &c.) where *Filaria bancrofti* is common, but where *Filaria loa* does not occur, supports the view that the former parasite is the causative agent.]

G. C. L.

WISE (K. S.) & MINETT (E. P.). Report of Tropical Diseases Research in the Government Bacteriological Laboratory, British Guiana, for the Six Months October, 1911, to March, 1912.—*Report of the Advisory Committee for the Tropical Diseases Research Fund for the Year 1912.* pp. 108-114. 1913. March. London: H.M. Stationery Office. [Cd. 6669.]

#### *Filarial Abscess.*—

Reference is first made to MAXWELL's work published in the *British Medical Journal*, Sept., 1901, pp. 609-612. In his paper abscesses of the scrotum, limbs and of the abdomen and thorax were described. Out of 23 cases recorded, however, remains of adult *Filaria bancrofti* were found in one abscess only and so MAXWELL was inclined to consider that some might be due to other causes than death of the parent worm, especially as regards abscesses of the scrotum. In all but the two last of his series filarial embryos were found in the peripheral blood at night.

Wise and Minett believe that the best evidence of these abscesses being filarial is undoubtedly the discovery of the adult worm in the pus. They therefore studied a series of deep-seated abscesses. These when opened were thoroughly washed out and all the contents completely evacuated, every drop of fluid and every shred of tissue being carefully collected. The whole of the material from each abscess was passed through a sieve of fourteen meshes to the inch and the resultant solid matter was washed thoroughly and teased out to find the worms. In several instances, more especially when the abscess was large, or of long standing or with much pus, no doubt the worm was absent because it had been destroyed by proteolytic digestion. Bacteriological examination of the pus was also carried out. The results are given in a table. Of 28 cases examined in 22 complete worms or pieces of worms were found. The authors believe that in the other six greater care or an earlier evacuation of the pus would have discovered the worm, and they have therefore no hesitation in saying that the adult *F. bancrofti* is a causative factor in these deep-seated abscesses. In 17 of the cases the abscess was the first pathological effect of the infection with *F. bancrofti*. In 7 instances filarial embryos were found in the pus and detritus removed from the abscess. As regards the bacteriology 21 abscesses showed streptococci in pure culture, four *Staphylococcus pyogenes aureus*, while three were sterile. It is pointed out that the death of an adult *F. bancrofti* does not necessarily lead to a filarial abscess, as has been shown by Wise in British Guiana and later by BARR in Fiji. The death of the worm frequently leads to its calcification. The recurrence of these abscesses in the same patient is a common feature, several of the cases showing four or five scars indicative of previous

attacks. One patient who subsequently died of heart failure, had had seven abscesses, three in the calf of the leg and four in the thigh, one after the other in quick succession. In conclusion the authors state that it is probable that the abscesses of the spermatic cord described by MAXWELL and the Endemic funiculitis of CASTELLANI are similar conditions due to the *F. bancrofti*. So far they have not found genuine filarial abscesses, such as are seen in the limbs, in the abdomen or thorax.

G. C. L.

WISE (K. S.) & MINETT (E. P.). Report of Tropical Diseases Research in the Government Bacteriological Laboratory, British Guiana, for the Six Months April, 1912, to September, 1912.—Report received in Colonial Office, Jan. 21, 1913.

*Septicaemia and Filaria bancrofti*.—

Reference is first made to previous reports relating to the connection between *Filaria bancrofti* and deep-seated abscesses in the limbs, and also to septic processes concerned with the lymphatics of the abdomen and thorax.

The exact nature of the relationship of *F. bancrofti* to septic processes such as abscess and septicaemia is a very interesting one. The condition which the authors now draw attention to is one of severe and extensive infection of the retro-peritoneal and retro-thoracic lymphatics. These lymphatics from the pelvic cavity to the region of the neck are filled with purulent matter containing streptococci of virulent character. Suppurating offshoots passing to the spermatic cord and testis, or to the glands in the groin, or by way of the axilla to the upper arm are also found.

Clinically, chilly feelings or definite rigors with pain in the abdomen form a common type of onset; occasionally rigor follows rigor in fairly rapid succession, though usually nothing more than the chilly ague fit is felt. The pain is not intense, nor does it cause much distress; usually when present it is greatest below the umbilicus and is increased by movement or pressure. The abdominal muscles are not hard and board-like but usually flaccid. The abdomen rarely becomes distended, tense, or tympanitic, because the peritoneal inflammation is usually limited to the posterior parts of the cavity. Vomiting is an early feature and sometimes occurs before peritonitis can have developed. Headache with delirium may also be seen. Death occurs in from two to eight days.

At the autopsy it is evident that the condition frequently follows suppuration of groin, axillary or cervical glands, filarial fever with lymphangitis of the leg or arm, suppurative lesions of the scrotum and cord, or deep-seated abscesses of the leg or arm.

The pathological anatomy is of a very peculiar and striking character. While certain parts of the pathological picture depend upon the particular area in which the suppuration commences, the central area to which all sooner or later converge is the retro-peritoneal or retro-thoracic lymphatics. Whether the inciting lesion is in the leg, in the epididymis, in the obturator region, in the groin glands, or elsewhere, the subsequent course is a slow progression of suppurative processes along the intervening

lymphatics to a final culminating infection of the retro-peritoneal or retro-thoracic lymphatics. An obvious mass of dilated lymphatics can be felt closely applied to the anterior and lateral aspects of the spinal column, and in this mass greatly hypertrophied lymphatic glands can be found. It may be impossible to trace any definite vessels but the main thoracic duct may usually be recognised. The vessels are all extremely thin-walled and markedly sacculated. The contents are either a brick-red fluid, reddish-white clot, or ordinary pus. In the thoracic duct the clot frequently becomes a pure white, more especially at the upper ending of the mass. In three out of fourteen cases *F. bancrofti* or its embryos were found in the abdominal mass. In each case the worms were discovered about the level of the kidneys. In no case was the adult filaria found in the thoracic duct obstructing the lymphatic flow at the upper end of the mass.

Smears of the material in the lymphatics and glands showed an enormous abundance of streptococci, and cultures bore out the extraordinary profusion of these organisms. Bacteriological examinations of thirty cases were carried out, and streptococci were observed microscopically in 28. In twenty instances they were isolated in pure culture and gave the characteristics of the ordinary *Streptococcus pyogenes*.

Stress must be laid, according to the authors, on the fact that the abdominal condition is a secondary one and that in every case with one exception the mass was connected with a similar lesion outside the abdominal cavity. The site of these has been described above.

The authors draw attention to somewhat similar cases described by DANIELS and CONYERS, by LOW as fever and ague of the abdomen, by MAXWELL and by MANSON, and conclude that the adult filariae are the exciting cause of the condition. Other observations and experiments in British Guiana show that while the death of adults of *Filaria bancrofti* may not infrequently lead to abscess when situated in the limbs, spermatic cord, or epididymis, the death of this worm within the abdomen or thorax rarely or never results in such a condition. The most usual sites in which the worms are found in the abdomen are the lymphatic tissues about the aorta and vena cava, the lymph spaces beneath the epithelium of the pelvis of the kidney, the lymph spaces of the sheath of the ilio-psoas muscles and in Glisson's capsule. It is probable that they may wander to all parts of the abdomen but they are certainly most readily detected in the above areas. When found they are either alive or, if dead, in a process of calcification, which proceeds until the worm has shrunk to a small coiled, calcareous nodule, about the size of a millet seed. The natural ending of the parasite would therefore seem to be death with calcification. In the course of the last few years the authors have found filariae either living, calcified, or undergoing disintegration in an abscess, in the lymphatics of the following situations:—The pelvis of the kidney (31 times), the epididymis (18 times), the retro-peritoneal tissues (12 times), the ilio-psoas muscles (4 times), Glisson's capsule (twice), inguinal glands (25 times), lymphatic vessels (8 times).

[There seems to be very little doubt that the filaria is the exciting cause of the condition just described. Suppurative lesions were also found to be very frequent by BÄHR in filarial cases in Fiji. The parasite, in some way or other, damages the lymphatics and so produces a condition favourable to the invasion and growth of micro-organisms.]

G. C. L.

SCHUMACHER. Eitrige Funikulitis. [Purulent Funiculitis.]—  
*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Jan. Vol. 17.  
 No. 1. pp. 18-19.

The patient was admitted into hospital complaining of pain in the right inguinal region and right testicle. His temperature was 39.2° C. The spermatic cord on the right side was thickened and swollen as was also the testicle. The swelling was very sensitive to pressure, and the skin over it showed commencing inflammation. The patient had had two similar attacks previously, a month and six months before. Schumacher operated and found a gelatinous and mucoid condition of the cord like that seen in chronic elephantiasis, with in addition acute inflammatory changes and multiple abscesses, the latter being found in the testicle also. The testicle and the cord were removed, healing was rapid, by first intention and the patient left hospital twenty days after the operation. In discussing the etiology of the condition the author suggests gonorrhoea as the cause.

[A similar condition has been described by CASTELLANI in Ceylon under the name "Endemic funiculitis." It is very likely that *Filaria bancrofti* is the real cause, ordinary cocci bringing about the suppuration as is so often seen in other filarial lesions.]

G. C. L.

## ELEPHANTIASIS.

GRAVOT. Un Cas d'Éléphantiasis complet du Scrotum et du Fourreau. (Oschéotomie avec Anaplastie.)—*Ann. d'Hyg. et Méd. Colon.* 1912. Oct.-Nov.-Dec. Vol. 15. No. 4. pp. 868-872.

A description of an ordinary case of elephantiasis of the scrotum with the operation for its removal is given. The disease is not common in the province of Fort-Dauphin, Madagascar, where the case occurred, though other forms of filariasis such as hydrocele and haematocele are frequently seen.

Haemorrhage proved troublesome at the operation. The wounds healed slowly and, owing to indiscretions on the part of the patient, signs of the condition relapsing became evident during his last fifteen days' stay in hospital.

G. C. L.

MÜLLER. Zur Operation der Elefantiasis der männlichen Genitalien. [On Operation for Elephantiasis of the Male Genitalia.]—  
*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Apr. Vol. 17.  
 No. 8. pp. 269-272.

The author describes the operative technique adopted by him in cases of elephantiasis of the scrotum and penis. The day

before the operation the parts are suitably cleansed and next day, twenty minutes before and again just before the commencement of the operation, are painted with ten per cent. official tincture of iodine.

A hollow sound is first introduced into the sac of the foreskin and a long incision is made in the middle line dividing the tissues covering the glans. The incision is now lengthened along the course of the penis, the proximal part of which is freed and then sound skin as far as possible is brought into relationship with it in order to form a covering.

The search for the testicles and their separation from the swollen mass is accomplished by a single oval incision. The upper pole of the oval coincides with the upper angle of the wound, the lower lies in the raphe of the perineum, as far removed from the anus as possible. The long aspects of the oval pass on the sides of the scrotum, encircling, so to speak, the tumour mass. They are then carried downwards to the depth of the testicles and cord, these latter structures, after being dissected out, being wrapped up in saline compresses for the time being. The tumour mass is then removed, bleeding points are stopped as far as possible, the incisions are united,—parts where this is not feasible being allowed to granulate—and drainage is provided for.

Three patients have been operated upon by this method with very satisfactory results.

G. C. L.

**BARNES (Noble P.). Filariasis; Report of Case Treated with Dioxydiamidoarsenobenzol.**—*Monthly Cyclopedic & Med. Bull.* 1913. Jan. Vol. 6. No. 1. pp. 1-7. With 4 figs.

The author describes a case of elephantiasis treated by salvarsan. Two injections were given, the second three months after the first. From the photographs illustrating the paper it would seem that the condition had been greatly benefited.

[Why this should be is not clear and as meagre details only are given it is impossible to draw any conclusions from them. The statement that microfilariae were found in the patient's stools is manifestly erroneous.]

G. C. L.

**ACOSTA-SISON (Honorio). Elephantiasis Glabra Congenitus with Report of a Case.**—*Bull. Manila Med. Soc.* 1912. Dec. Vol. 4. No. 12. pp. 196-198.

Congenital elephantiasis is a very rare condition, so the report of the above case is of great interest.

The child, a male, was born healthy and robust, but with the left lower extremity from Poupart's ligament to the ankle joint, including the buttock, very much enlarged. The skin of the affected extremity was somewhat indurated and did not pit on pressure. Two years later the hypertrophy of the left lower extremity had slightly increased and had extended upwards to the left side of the lower abdominal wall and downwards to the



dorsum of the left foot. The glans penis was also much enlarged and had reached the size of a large cherry.

Three possibilities, according to the author, must be considered in the diagnosis of the affection, viz.:—(1) lipomatosis, (2) obstruction of the left common iliac vein, and (3) obstruction of the lymphatics in the region of the left common iliac glands.

In lipomatosis, however, the hypertrophy is usually bilateral and is usually greater in regions where there is normally an abundance of fatty tissue. In obstruction of the left common iliac vein, the oedema is usually soft and the superficial veins are dilated. Such being the case, the condition was evidently due to some obstruction in the lymphatics, but what the nature of that was could not be determined. In ordinary acquired elephantiasis the most common variety is that which results from lymph stasis produced by any factor that causes obstruction to the lymph vessels, such as *Filaria bancrofti*; chronic lymphangitis; erysipelatous or eczematous inflammation of the skin involving the lymphatics; lupus; syphilis; varicose ulcers; frost bite; traumatism, etc. Removal or destruction of the regional lymph glands has also been known to cause elephantiasis of the part tributary to the glands. The parts may be hard and indurated (elephantiasis dura), soft and pitting on pressure (elephantiasis mollis), the epidermis may be smooth (elephantiasis glabra), papillary or warty (elephantiasis verrucosa) or nodular (elephantiasis tuberosa). The legs and external genitals are most frequently involved though the disease may occur in any superficial part of the body.

[A subsequent history of this interesting case would be of value.]

G. C. L.

BEVACQUA (Alfredo). *Fuso-spirillare Assoziation in einem Falle von Pseudo-elephantiasis des unteren linken Gliedes bei einem Araber.* [Fuso-spirillar Association in a Case of Pseudo-elephantiasis of the left lower Limb in an Arab.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Mar. 1. Vol. 68. No. 2. pp. 182-188. With 3 figures.

The case was seen in Tripoli. The condition was a chronic one, having lasted for years and was said to have followed an injury to the foot. Bacteriological investigations showed many organisms, amongst these spindle formed bacilli somewhat similar to the fusiform bacillus of VINCENT, and in addition numerous spirilla, with two or three spirals, resembling the *Spirochaeta refringens* found in the mouth. There were no signs of leprosy or tubercle bacilli. A photograph of the affected limb, a microphotograph of the organisms and one of a section of the tissues are given. In the latter giant cells appear to be present. [The bacilli present are probably only secondary contaminants and have nothing to do with the original cause of the disease.]

G. C. L

DANIEL (C.). **Die Elephantiatische Tuberkulose der Vulva.** [Tubercular Elephantiasis of Vulva.]—*Monatsschr. f. Geburtshilfe u. Gynäkologie.* 1913. Jan. Vol. 37. No. 1.

A case of this rare condition is described while seven others are quoted from the literature. In coming to a diagnosis other causes of elephantiasis, such as filariasis and syphilis, have to be excluded. If the patient has never lived in the tropics filariasis may be put out of court. The author recommends excision for the treatment, but in the majority of cases the disease recurs and general tuberculosis may appear.

G. C. L.

SÉZARY (A.) & SALÈS (G.). **Eléphantiasis bacillaire.**—*Rev. de Médecine.* 1913. Feb. 10. Vol. 33. No. 2. pp. 111-115.

A non-tropical case of elephantiasis is described. [These cases though rare, crop up occasionally. See this *Bulletin*, Vol. 1, pp. 93 and 94.] The condition was a unilateral one and followed chronic tubercular disease of the knee, other signs of tubercle being present in the lungs and elsewhere. The authors therefore classify their case as one of bacillary elephantiasis. [Any chronic infection, especially if associated with extensive fibrosis, may block the lymphatics and so cause elephantiasis.]

G. C. L.

#### DRACONTIASIS.

BOUILLIEZ (M.). **Orchite due à la Présence d'un Ver de Guinée dans le Testicule.**—*Ann. d'Hyg. et de Méd. Colon.* 1912. Vol. 15. No. 4. pp. 872-875.

The clinical details which accompany the record of this case illustrate very strikingly the difficulties of diagnosis which may present themselves where a large parasite like the guinea-worm is traversing deep seated and vital organs and the permanent damage which may follow upon such wanderings.

The patient, a sergeant in the French Colonial Infantry, was suddenly taken ill with fever and with pain in the abdomen, the cord and the left testicle. The cord was hard and very painful. Micturition was frequent but painless, the urine contained a large quantity of pus but no blood. Drops of pus came away in the intervals of micturition. The fever, with nausea and vomiting, increased for the succeeding two days; then the urine cleared and no longer contained pus; a little could, however, be obtained at the meatus by pressing along the course of the urethra. At the same time the cord had become less hard and painful, but the left testicle had enlarged to three times its normal size. When the patient was seen by Bouilliez, seven days after the onset of the illness, the fever had completely subsided: there was no vomiting or nausea. The urine was clear and the urinary meatus dry. The testicular cord had regained its normal consistence, but was still tender. The testicular mass was swollen, hard and very painful. One spot, above and below, seemed slightly softened but neither more nor less painful than elsewhere.

The usual treatment for orchitis: an ointment of mercury and belladonna; light compression by means of wad-dressings; a suspensory bandage, etc., was adopted. This treatment greatly relieved the symptoms, although the testicular condition remained practically unchanged. After fifteen days, accompanying intense itching, a small vesicle formed at the soft spot noted previously in the testicular mass and a guinea-worm appeared. By slight traction this was extracted and was found to be sixty cm. long. The tiny wound healed in three days.

Following upon the extraction of the guinea-worm the testicle began to atrophy and five months later scarcely exceeded a large haricot bean in size. The author discusses, in the form of a series of interesting questions, the probable course followed by the guinea-worm from the neck of the bladder to the interior of the testicle.

R. T. Leiper.

ROUBAUD (E.). *Observations sur la Biologie du Ver de Guinée: Infection intestinale des Cyclops.*—*Bull. Soc. Path. Exot.* 1913. Vol. 6. No. 4. pp. 281-288.

This memoir contains several important additions to our recent knowledge of the bionomics of the guinea-worm and its development in *Cyclops*.

*Survival of embryos.*—In no instance did guinea-worm embryos survive for longer than five days. Taken fresh from the female they only lived three days in water and four days in moist earth in Dahomey when the mean temperature was 25° C. Nor could any appreciable capacity of resistance to desiccation be demonstrated.

*Manner of infection of Cyclops.*—Roubaud discards the generally accepted view that the guinea-worm embryos infect the *Cyclops* by actively penetrating into their bodies through the cuticular skin and maintains that he has experimentally confirmed the view hitherto held alone by LEIPER that the *infection is intestinal*. FEDTSCHENKO stated that embryos ingested by the *Cyclops* were killed by the stomach juices, but Roubaud affirms that the *Cyclops* swallow dead as well as living embryos.

The following experiment is detailed in support of the theory of intestinal infection. A large number of *Cyclops* were placed in water rendered cloudy with embryos. Six hours later the bulk of the *Cyclops* examined showed voluminous groups of embryos in the region of the stomach. The worms were active and did not by any means appear to be digested. In the fresh material no embryos could be detected in the general body cavity at this stage, but in sections some embryos can be seen already, in some instances, in the body cavity alongside of the stomach group. In those *Cyclops* examined after 24 and 48 hours no embryos could be found in the digestive tract: all had passed into the body cavity. If the embryos had been digested one would have expected to find traces of cuticle in the intestinal content.

Another new observation of interest is that the nauplius larva of the Cyclops is also susceptible of infection. Roubaud states that to find three or four embryos in a single nauplius is not rare.

*Ecdysis.*—In Dahomey the first sheath forms on the eighth day. This is a minimal period for even on the tenth day one may find even in the same Cyclops some embryos which have their primitive characters. By the fourteenth day the larvae have assumed the appearance of the second stage. The sharp tail of the new born embryo has given place to one short and blunt. The striation of the cuticle is much less apparent but still visible. The larva is cylindrical and now measures 0.5 mm. The movements are much less rapid than in the first stage. Larvae 42 days old show the following features. Size has increased to 0.6 mm. The mouth has two distinct lips. The posterior third of the body is now distinguished by a yellowish brown colouration [due doubtless to pigmentation of the wall of the intestine]. The early mobility has completely disappeared and they lie coiled up in their host. No terminal papillae could be made out at the posterior end of the body. The tail is simply conical not trilobed.

*Infection experiments* were made upon three monkeys (*Cercopithecus viridis*) with (1) embryos taken fresh from the uterus of a mature worm, (2) a dozen Cyclops infected 48 hours previously, and (3) fifteen Cyclops infected for a fortnight. None of these were found infected at the autopsies made several months later. No experiments were made with Cyclops infected during a period of six weeks.

*Seasonal incidence.*—In Dahomey dracontiasis is most frequent from December to February, the driest months of the year. The persistence of endemic dracontiasis seems to be the result of a complex equilibrium between (a) the time necessary for the annual development of the worm in man, (b) the regular periodic recurrence of the seasonal conditions, and (c) those conditions of human existence which favour its transmission by the Cyclops. The geographical distribution of the guinea-worm is determined by the local fulfilment or otherwise of these three essentials.

R. T. L.

#### UNCLASSIFIED.

DEGORCE (A.). *Le Traitement Chirurgical du Varicocèle Lymphatique.*—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. Jan. Vol. 4. No. 1. pp. 39-45.

Lymphatic varicocoele is comparatively rare in Tonkin, Degorce only having had occasion to operate in three cases in the course of ten years' hospital practice in Hanoi. During that time a greater number of cases were seen, but these were either unsuitable for operation, or the patients refused surgical treatment. The results, in the three cases mentioned above, were not very brilliant. In one, who suffered from a double lymphatic

varicocele, extirpation was performed on the right side, but this was followed by the onset of chyluria and a bad general state. In another, some days after the operation, the lymphatics of the testicle dilated, though this symptom had not been present before the excision, while in the third the scrotum remained infiltrated, oedematous, and tender to pressure. As Degorce himself says, these dilated lymphatics are only a part of a general dilatation which may extend up on the posterior aspect of the abdomen as high even as the thoracic duct, and therefore their removal cannot in any way influence the cause of the condition. Unless the patients are suffering great pain and have hydrocele and hernia as well, ROY DES BARRES believes the condition is best left alone. As complications that observer has noted two cases of lymphorrhagia. CLARAC has also drawn attention to the danger of producing haematochyluria after such operations.

The technique of the operation itself is simple. Degorce and ROY DES BARRES open the inguinal canal and resect the dilated lymphatics from the superior extremity of the testicle to the internal ring. Hernia, if present, is dealt with and hypertrophied lymphatic glands are removed. The spermatic artery can be recognised by its pulsation and red colour, the veins are bluish, while the lymphatics are grey and of a doughy consistence. In some cases the artery can be easily isolated from the other constituents of the cord, but in others it may be practically impossible to separate it and then it may have to be ligatured. In the latter case, however, there may be a danger of consequent atrophy of the testicle. If hydroceles exist they should be dealt with by resection of the tunica vaginalis and if the scrotum is soft and baggy (lymphscrotum) it may be partially resected and so reduced in size. After this the inguinal canal is reconstituted by suturing, drainage being allowed for in some cases, in others not.

G. C. L.

BLUEL (Adolphe). *Contribution à l'Etude de la Filariose de Demarquay.*—Université de Bordeaux. Thèse pour le Doctorat en Médecine. (No. 110.) 1912. 66 pp. Bordeaux: Imprimerie Barthélemy & Clèdes.

The author bases his thesis on a case of ordinary filariasis, lymphangitis of groins, and orchitis, from Guadeloupe. In addition to these manifestations of *Filaria bancrofti* infection, the patient had embryos of *F. demarquayi* in his blood [i.e. suffered from a double infection]. Frequent attacks of lymphangitis were complained of these occurring every few months. Salvarsan was given and since the injection of this drug no further attacks had appeared up to eleven months afterwards. The author gives a description of *F. demarquayi* in which he quotes largely from the writings of DANIELS, Low and others on the subject. [The patient came to France from Guadeloupe to finish his education. If it should turn out that he had never been out of that island before, a fact which is not stated, this would mean that Guadeloupe is

another West Indian Island in which *F. demarquayi* is endemic. This important point is not brought out in the thesis nor the apparent fact that the patient was suffering from a double infection.]

G. C. L.

KING (Arthur). **A Case of Filariasis in Devonshire.**—*Brit. Med. Jl.* 1913. May 24. p. 1108.

The author believes that he has discovered a filaria in the vaginal discharge and blood of a girl who had never been out of Devonshire.

[No details are given. Such discoveries have been made in England before but have never been confirmed.]

G. C. L.

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## MISCELLANEOUS.

- i. SCOTT (H. Harold). **Fulminating Cerebro-Spinal Meningitis in Jamaica.**—*Ann. Trop. Med. & Parasit.* 1913. Mar. 31. Series T. M. Vol. 7. No. 1. pp. 165-181.

**A Report by the Government Bacteriologist on Cases of Cerebro-Spinal Meningitis, indicating the Similarity between the Symptom-Complex of that Disease and some Cases of Vomiting Sickness.**—7 pp. F'cap. 1913. Jamaica: Government Printing Office, Kingston.

- ii. POTTER (T. J.). **Report on Vomiting Sickness in Jamaica.**—*Report of the Advisory Committee for the Tropical Diseases Research Fund for the Year 1912.* Appendix vii. pp. 172-198. 1913. London: H. M. Stationery Office.

i. A disease known by the name of vomiting sickness prevails in Jamaica. Scott, the Government Bacteriologist, suggests that it is really nothing more nor less than fulminating cerebro-spinal meningitis. Several official reports have already appeared on the subject. On the 4th September 1912 a sudden outbreak of a very acute nature occurred in Kingston itself and these cases were specially investigated. The following is the author's summary of what he found:—

"1. In four cases presenting the typical symptoms of 'vomiting sickness' the Weichselbaum's diplococcus has been isolated.

"2. These symptoms correspond to those associated with foudroyante types of cerebro-spinal meningitis.

"3. The organism was obtained from the cerebro-spinal fluid of all those from whom this fluid was taken during life, and from the cerebral ventricular fluid post mortem.

"4. The organism was obtained from the naso-pharynx of one of the contacts of these cases, though himself apparently in perfect health.

"5. The intracranial post-mortem signs were such as are consistent with death from cerebro-spinal meningitis at such an early stage, that is, with fulminating cases of the disease."

"This focus did not spread, and the child who was proved to be harbouring the organism in his naso-pharynx did not develop the disease."

Later, in October, 1912, more cases occurred in Kingston, and these also were specially studied. The general conclusions arrived at are as follows:—

"1. In several cases of illness with sudden onset in apparently healthy subjects, terminating fatally in a high percentage of those attacked, I have isolated a Gram-negative diplococcus from the spinal fluid.

"2. This organism gives the morphological and cultural characters of Weichselbaum's *Diplococcus intracellularis meningitidis*, except that in galactose it does not always give a definite reaction, the medium in some instances not being affected. The maltose and dextrose, however, are always typically acted upon.

"3. These cases exhibit in many instances a symptom-complex which has for years been spoken of in Jamaica as 'Vomiting Sickness.'

"4. The disease breaks out in localised epidemic form in various parts of the island every cold weather, that is, from about the middle of December to the end of February or beginning of March.

"5. The disease occurs at other times as sporadic cases, but apparently does not spread extensively.

"6. It is a curious thing that practically all the cases are of the hyperacute variety, either recovering in a few days, or dying in a few hours. One rarely sees the subacute cases during the epidemic times, and never, so far as I am aware, the chronic ones, unless some of the natives one meets with who are chronically deaf, or have seriously defective vision, or who suffer from fits, are instances of cerebro-spinal meningitis with permanent sequelae. Of this there is no proof.

"7. In two instances a spirochaete-like body, or long curving bacillus, has been seen in the smears made directly from the fluid obtained by lumbar puncture. What part, if any, this takes in producing the symptoms I have had no means of discovering.

"8. The disease is very rare amongst the white population; I have known of only three instances so far during the past year. Possibly this is due in part to better hygienic conditions, less crowding, and so forth, but not entirely, because

"9. I have not personally met with the condition once amongst the East Indian population, where overcrowding and bad hygiene are nearly, if not quite, as marked as in the case of the native. For this, also, I am unable to offer any explanation at present, though it may be noted that the food supply of the East Indian is usually better than that of the West."

ii. Potter, in discussing the subject, states that the condition diagnosed as cerebro-spinal meningitis in the past is the same as that now diagnosed as vomiting sickness. Places where cerebro-spinal meningitis, occurring almost exclusively among young children with a very high case mortality, used to appear are exactly those in which the so-called vomiting sickness is now got. He believes that had these cases been cerebro-spinal meningitis some would have run a more prolonged course, sequelae such as paralysis, blindness and deafness would have followed, and post mortem examinations would have shown definite inflammatory changes in the meninges.

Ptomaine poisoning, ackee poisoning, and cassava poisoning have all been at various times recorded as the cause of vomiting sickness.

The author then tries to show that up to the year 1904 yellow fever was looked upon as endemic in Jamaica, and post mortems made by himself as well as those collected from reports leave no doubt in his mind that it is still endemic. He states that it is significant that one of the popular alternative names for vomiting sickness is black vomit and that it is remarkable that in countries where yellow fever is admitted to be endemic—Brazil, Peru, and Mexico—there is a disease described, in every way similar to the vomiting sickness of Jamaica. Children are mainly attacked, the onset is sudden, vomiting is a prominent symptom, and the vomit is frequently haemorrhagic, sometimes black. The case mortality is high and in each country there exists the same desire to avoid calling the disease yellow fever. Potter also considers it of interest that, among all the explanations of the cause of vomiting sickness, yellow fever has never been suggested, and this in spite of the fact that one of the popular terms for the disease is black vomit.

An appendix gives a list of the cases seen, 70 in number, and also of the post mortems made. The conclusion is that there is no justification for the use of the term vomiting sickness. The disease is not a separate clinical entity and the author is of opinion that the majority of the deaths ascribed to it are due to yellow fever.



[It is not quite easy to reconcile the definite conclusions reached by Scott and Potter, unless one believes that both epidemic cerebro-spinal meningitis cases and yellow fever ones have, in the past, been mistaken for vomiting sickness. Certainly some of the post mortems of Potter's cases are extremely suggestive of yellow fever, while similarly there seems to be no reasonable doubt about the diagnosis of epidemic cerebro-spinal fever in Scott's cases.]

G. C. Low.

LÖHLEIN (M.). *Beiträge zur Pathologie der Eingeborenen von Kamerun. iv. Übersicht über die Ergebnisse von 140 Sektionen in Duala 1910 und 1911.* [A Summary of the Results of 140 Autopsies in Duala, Cameroons, 1910 and 1911.]—*Beihefte zum Arch. f. Schiffs- u. Trop.-Hyg.* 1912. Nov. Vol. 16. Beiheft 9. pp. 91-109. [727-745.]

The principal diseases which caused death in the 140 cases were as follows:—

	No. of cases.		No. of cases.
Abscess of Liver	... 4	Malaria	... 7
Aneurism	... 1	Meningitis	... 6
Ankylostomiasis	... 4	Peritonitis	... 1
Cirrhosis of Liver	... 4	Pneumonia	... 28
Beriberi	... 2	Sarcoma	... 1
Bronchitis	... 1	Sleeping Sickness	... 3
Endocarditis	... 2	Smallpox	... 2
Enteritis	... 1	Surgical	... 12
Dysentery	... 28	Tubercle	... 12
Leprosy	... 1	Typhoid	... 10

Various:—Suicide, Cellulitis of leg, Suppurative Parotitis, Noma, Hyperaemia and Oedema of lungs (two cases), Purulent arthritis, Rupture of spleen (two cases).

Examinations made at the autopsy for animal parasites showed that ankylostomes, trichocephalus and other intestinal parasites were very numerous. In addition, porocephaliasis was found to be not infrequent, ten cases being noted in the above series.

From the table it will be seen that dysentery and pneumonia were the most common cause of death; typhoid, tubercle and malaria coming next in frequency. [Interesting statistics of post mortems such as the above should prove of value to workers in the tropics.]

G. C. L.

MASTERMAN (E. W. G.). *Notes on some Tropical Diseases of Palestine.*—*Jl. of Hygiene.* 1913. Apr. Vol. 13. No. 1. pp. 49-62. With 1 chart.

Malaria is very common in Jerusalem. The Anopheline host breeds in the numerous cisterns throughout the town. MÜHLENS states that this is the *Anopheles bifurcatus*. CROPPER (1902) has shown that four species of Anophelines are found in Palestine,

viz.:—*Anopheles maculipennis*, *Pyretophorus palestinensis*, *Myzorhynchus pseudopictus* and *Cellia pharoensis*.

Haemoglobinuric (blackwater) fever is now well known in Palestine. It cannot be called common in Jerusalem, but every autumn a few cases occur amongst patients who have not been outside the city for years. In the maritime plain, around Jaffa, and in the malarious regions near Caesarea, it is far from uncommon. The worst region of all is the Jordan Valley near Lake Huleh and south of the Sea of Galilee, in both of which places many people have succumbed and others have been reduced to states of extreme anaemia.

Dengue prevails throughout Palestine. During the autumn months of 1912 a severe epidemic raged in Syria. The last epidemic in Jerusalem occurred in 1889, but in Jaffa and along the coast the disease is frequent. In making the diagnosis of dengue two other diseases, besides malaria, have to be excluded, viz.:—influenza and three-day fever. The author states that GRAHAM'S original idea that the carrier of the disease is a mosquito is a correct one, but he does not know of any evidence that proves that *Culex fatigans* is necessarily the only carrier.

[Other tropical conditions in Palestine would appear to be rare; at least, they are not mentioned in the article.]

G. C. L.

SANTAMARIA (J. Martinez). **Some Notes on Tropical Diseases observed in the Republic of Colombia.**—*Jl. Trop. Med. & Hyg.* 1913. Apr. 1. Vol. 16. No. 7. pp. 100-102.

The author writes a short note upon certain tropical diseases found in Bogota, Colombia. These are similar to those found in other parts of the world; for example, yaws, pinta, leprosy, yellow fever, relapsing fever, malarial fever, blackwater fever, tropical splenomegaly, beriberi, dysentery, ankylostomiasis and other forms of helminthiasis, piedra, etc. The Spanish name of these with a short description of each is given.

G. C. L.

WHITMORE (A.). **An Account of a Glanders-like Disease Occurring in Rangoon.**—*Jl. of Hygiene.* 1913. Apr. Vol. 13. No. 1. pp. 1-34. With 1 plate and 2 charts.

Some of the work in this paper has already been referred to (see this *Bulletin*, Vol. 1, p. 588). The author now goes into the subject in greater detail and gives a short account of 38 cases in which he has isolated his glanders-like bacillus. The pathological lesions are also described, patches of consolidation in the lungs being one of the principal of these. The lesions are often so peculiar in appearance, as at once to suggest that none of the usual pathogenic bacilli could have been responsible for their production. Further it is stated that if guinea-pigs be fed with food or drink contaminated with pure cultures of the new bacillus the animals speedily die, and after death, not only can the bacilli be easily recovered from their organs, but also lesions are present in every way similar to those found in the human subject.

The human disease is characterised by the presence of the bacillus and the definite lesions. Its nature is that of a pyaemia or septicaemia clinically resembling glanders, but according to the author the distinction between the two diseases is easy if a proper bacteriological examination is carried out. Confusion may arise if Strauss's guinea-pig testicular reaction is relied upon (*loc. cit.*).

Clinically, knowledge of the disease is scanty and only a suspicion of the infection can be entertained on clinical signs alone. It seems to be specially prevalent amongst morphia injectors.

G. C. L.

**Investigations of Rocky Mountain Spotted Fever.**—*Annual Report of the Surgeon-General of the Public Health Service of the United States for the Fiscal Year 1912.* pp. 28-30. 1913. Washington: Government Printing Office.

In the Report for 1911 a description was given of investigations on Rocky Mountain spotted fever carried out from May 12 to August 7, 1911. Passed Asst. Surg. T. B. McCLINTICK began these studies again on April 1st, 1912, and, as in the previous year, the methods adopted were:—(1) Tick extermination by (a) dipping of domestic stock and (b) killing of small wild animals; and (2) Laboratory investigations. From April 1st to July 1st, 1912, 402 horses, 851 cattle, and 157 sheep were dipped. It is proposed for the future to dip the stock early before the ticks appear and then afterwards once a month.

War was waged against small mammals, the commonest of which was the Columbian ground squirrel, *C. columbianus*. In addition to this species other small wild mammals, such as pine squirrels, chipmunks and woodchucks are hosts for feeding and developing the larval and nymphal forms of the tick. Poisoning and fumigating with carbon bisulphide proved very efficient in the work of extermination.

In the laboratory, investigations were made to study the susceptibility and immunity of some of the wild mammals to spotted fever infections. These were inoculated with the virus of the disease and then about five days later inoculation of their blood was made into guinea-pigs. The mammals used were as a rule caught in territories badly infested with ticks. 175 ground squirrels, 20 woodchucks, 1 coyote, 12 rock squirrels, 1 mountain goat, and 1 wood rat were examined. The experiments have not yet reached the stage at which definite results can be reported. 150 ticks collected in the State of Idaho, where spotted fever is very prevalent in mild form, were placed on guinea-pigs, but none of the animals developed the disease.

At the close of the season McCLINTICK, unfortunately, became infected himself, presumably in his laboratory, and died of the disease after an illness of six days.

G. C. L.

PARANHOS (Ulysses). *Considérations sur le "Mal d'Engasgo."*—*Bull. Soc. Path. Exot.* 1913. Jan. Vol. 6. No. 1. pp. 47-50.

"Mal d'engasgo" or "entalação" is an endemic affection found in certain regions of Brazil. It is characterised by difficulty of swallowing, which may become so complete as to prevent the ingestion of food and cause inanition and death. The disease has been known for a long time in the states of Parahyba, Bahia, Minas, Goyas, Matto-Grosso, and Sao-Paulo.

What causes the obstruction is not quite clear. It may be due to a spasm of the lower part of the oesophagus preventing the passage of the bolus of food into the stomach. There is no organic stricture, as the oesophageal bougie passes freely into the stomach. Radioscopy reveals nothing. The oesophagus in some cases is dilated. The diagnosis is not difficult and the differentiation from organic stenosis and hysterical spasm is easy. The prognosis is always bad, the patients generally becoming cachectic and liable to intercurrent affections; the duration of the disease varies greatly, lasting for months or years. The treatment is purely symptomatic. Bromides, chloral, opium, and injections of iron and arsenic have been tried. The pathological anatomy has not been worked out, no autopsies apparently having been made. Some believe that the condition, for which the author proposes the name "*Dysphagie tropicale*," is essentially a neurosis.

BOUCHARD has stated that the symptoms of this disease as seen in Brazil are very similar to those of a disease of pheasants in France, due to the presence of worms in the oesophagus. Others suggest that the condition might be a clinical variety of the trypanosomiasis of CHAGAS, while others believed that the cause might lie in an intoxication by manioc. The prolonged ingestion of this substance seems to set up motor disturbances, dysphagia, convulsions, cardiac feebleness and death with arrest of respiration. In making flour from manioc the toxic principle is destroyed by the action of heat but if the process is insufficiently carried out enough might be ingested to cause toxic symptoms.

[More research on this interesting condition is manifestly required.]

G. C. L.

GABBI (U.). *La "Fièvre Boutonneuse" à Tripoli.—Malaria e Malat. d. Paesi Caldi.* 1913. Mar. Vol. 4. No. 2. pp. 65-68.

Attention is drawn to a febrile infection of unknown etiology, which is characterised more by its particular exanthem than by any special course. A complete and incomplete history of two cases is given. The clinical symptoms of both of these correspond closely to the descriptions given by CONOR and BRUCH of a disease in Tunis, to which the name of *Fièvre boutonneuse* has been given. This peculiar malady is characterised by:—

1. An eruption which appears on the trunk and limbs on the second or third day and consists of a red staining or mottling,

the spots being of the size of a lentil, often of unequal dimensions, and having an indurated base. These give the impression of being raised, but this is not borne out when they are touched, an observation first made by BANCHI. They are not painful, nor itchy, and may affect the palms of the hands or soles of the feet. After the fall of the fever they gradually disappear and leave a slightly pigmented trace behind.

2. An occasional exanthem of the buccal mucous membrane.

3. The temperature, which rises suddenly with rigors, sometimes associated with vomiting and cerebral phenomena.

4. Muscular and periarticular pains often of great severity.

The malady lasts from ten to fourteen days and is not accompanied by disturbances of the circulatory, respiratory or urinary systems. Relapses do not occur and the prognosis is a good one. Cases appear in the autumn.

Gabbi does not believe the malady is the same as Brill's disease, which is now generally supposed to be a form of typhus (*tifo esantematico*).

G. C. L.

SCORDO (F.) & RIZZUTI (G.). *Considerazioni Cliniche e Ricerche Batteriologiche in una Epidemia d'Ittero Infettivo a Tripoli*. [An Epidemic of Infectious Jaundice in Tripoli in its Clinical and Bacteriological Aspects.] — *Policlinico*. Sez. Med. 1913. Apr. Vol. 20. No. 4. pp. 145-170.

A widespread epidemic of pseudo-catarrhal jaundice which appeared amongst the Italian troops in Tripoli gave the authors the opportunity of conducting certain researches on the etiology of this disease. BLOIS has already proposed the name of infectious catarrhal jaundice but the authors prefer infectious pseudo-catarrhal jaundice. CHAUFFARD gives the clinical symptoms as follows: Sudden onset, headache, pains in the limbs, trunk, and neck, vertigo, loss of appetite, dirty tongue, nausea or vomiting, insomnia, extreme prostration. There is also fever oscillating from 38.5° C. to 39.5° C., epistaxis, labial herpes (about third or fourth day), bilious diarrhoea more or less prolonged, enlargement of spleen, slight enlargement and pain in liver, with a trace of albumin in the urine. About the fifth or seventh day jaundice appears; there is then a crisis with a fall of temperature and amelioration of the symptoms. The clinical signs found by the authors in their series of 25 cases corresponded closely with those just described. There does not appear to be any record of deaths occurring. Agglutination experiments were made and also bacteriological researches of the blood. A good number of the cases reacted to paratyphoid B. The results of haemocultures were negative as KUHN found in an epidemic of 58 cases, some of considerable gravity. The authors think, however, that the condition is due to a septicaemia of some sort or other, the infection descending by the biliary passages.

G. C. L.

**BRAULT (J.).** Note sur une Forme d'Adénites Subaiguës de l'Aine, rencontrée en Algérie.—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 201-202.

During twenty years as a surgeon in Algiers, Brault has specially noted a form of subacute adenitis of the groin. Clinically these swellings are typical and they have a special pathological anatomy. Pus occasionally forms and sometimes staphylococci or streptococci are found, but in other instances the collection is absolutely sterile. Inoculation of the pus and pieces of the tissue of the glands into guinea-pigs always gave negative results as regards tubercle, nor did sections of the glands show any evidence of tubercle bacilli or giant cells. Venereal and other causes could also be excluded. [Filariasis is not mentioned as a possible factor, but on the whole the account reads more like climatic bubo than anything else.]

G. C. L.

**BOWLES (F. H.).** Coccidioidal Granuloma. Report of the Nineteenth Case in California.—*Jl. Amer. Med. Assoc.* 1912. Dec. 21. Vol. 59. No. 25. p. 2253.

Up to the present time eighteen cases of coccidioidal granuloma have been reported and traced to California. Bowles now describes a nineteenth. OPHÜLS has described the fungus found in these cases under the name of *Oidium coccidioides*, differentiating it from the closely related *Blastomyces coccidioides* about which much has recently been written.

The duration of the disease is from six months to nine years. Bone lesions seem to be common, some of these having accompanying lesions like hypertrophic lupus. Tumours of the skin followed by ulceration without tendency to heal have also been noted.

The mode of infection, whether external or internal, has not been determined, but in the forms that produce a skin infection as the first symptom the skin is in all probability the primary focus. In internal cases infection is most frequently seen in the lungs. The infection travels by the blood and lymph streams. At autopsy the internal organs are seen to be more or less attacked with lesions similar in distribution to those of tuberculosis.

Most of the previous cases were treated with potassium iodide and surgically. All proved fatal with the exception of one in which the first symptom was a bone lesion of the ankle. In this instance the leg was amputated before other lesions developed and the patient is reported alive and well. The present case occurred in a Japanese farmer who had lived in California since coming from Japan in 1909.

The previous history was negative, venereal diseases being denied. During the year 1910 he was working in the San Joaquin valley as a teamster in a vineyard. In January, 1911, he was taken ill, treated for typhoid fever and was in bed for two months. During the latter part of this time tenderness developed in the right heel with slight swelling. After the fever abated the patient was permitted to be up; the tenderness and swelling remained. During the latter part of March, pain and tenderness developed in the left shoulder and later in the left hip. Work was

resumed, but in a few days he was compelled to stop on account of weakness.

In July the patient first came under observation presenting the following symptoms: He was fairly well nourished with the appearance of being ill, with pain, tenderness and swelling at the crest of the left ilium near the sacro-iliac joint and of the right heel; x-rays showing bone destruction in the calcis and ilium. There was slight swelling and tenderness also over the left scapula and over the second and third metacarpal bones of the right hand. Further physical examination proved negative except for slight tenderness along the colon.

Surgery was resorted to, incisions being made along the crest of the ilium and over the os calcis posteriorly; the necrosed bone was curetted and drained. A great deal of pus-like material escaped with the necrotic bone, which at this time gave no growth on agar; smears showed granular material without leucocytes or bacteria. The patient remained in hospital for six months, during which similar lesions gradually developed in the left scapula, right metacarpal bones, lower end of the left humerus and left tibia. These received the same operative treatment but showed no tendency to close in spite of after-treatment. A small nodule next developed on the forehead midway between the hair line and the glabella, which grew to be as large as a hazel-nut without tenderness or tendency to soften, being firmly attached to the bone, but not to the skin. There were occasional slight rises of temperature during the later part of the patient's stay at the hospital.

Cultures made from the lesions were positive, a fungus developing which later was identified by OPHÜLS as *Oidium coccidioides*. The patient passed from observation in 1912, having left for Japan. He died two months later.

G. C. L.

MAYER (T. F. G.). **A New Mosquito-proof and Storm-proof House for the Tropics.**—*Ann. Trop. Med. & Parasit.* 1913. Mar. 31. Vol. 7. No. 1. pp. 41-44. With 1 plate.

The author has had a house designed for him for use in the tropics, embodying certain principles which he believes are new.

The house, an illustration of which is given, is constructed almost entirely of steel, wood-work being reduced to a minimum. It is built on a plinth of concrete and has a floor and low wall of similar material. The walls are continuous with a steel framing which is filled in completely with mosquito netting made of a specially woven composite material. This netting is sandwiched between perforated metal sheets which prevent bulging and render entry impossible except by the spring doors at each end of the lobby entrances. The roof is completely shut off from the rooms below by an asbestos ceiling. Walls and partitions within the mosquito proofing are done away with in order that any breeze may blow straight through the house from one side to the other. Shutters, provided all round the house, can be shut if the wind be too cold or too strong. The division of the interior of the house is left to the individual occupant; it is suggested that by the use of sun blinds, screens and curtains the open character of the house may be preserved, but permanent brick or stone walls may be built if desired.

It is possible to make such a house of any number of stories and build it on piles if desired.

G. C. L.

MITZMAIN (M. B.). *Stomoxys calcitrans*, Linn. A Note giving a Summary of its Life-History.—*U.S. Public Health Rep.* 1913. Feb. 21. Vol. 28. No. 8. pp. 345-346.

During the two years' experience as Entomologist to the Bureau of Agriculture in the Philippine Islands the author has specially studied the development and life-history of *Stomoxys calcitrans*. The following is a summary of his study:—

"1. The age at which the female begins egg laying has been determined in bred flies as the ninth day.

"2. The maximum number of eggs produced by a single *Stomoxys* may be placed at, at least, 632 and possibly 820. As many as 20 depositions are made in the lifetime of a female. The maximum number of eggs deposited at one period was found to be 94.

"3. The incubation period for these eggs is 20 to 26 hours at a temperature of 29° C. to 31° C.

"4. The larval stage under favorable conditions is usually 7 to 8 days.

"5. The imago emerges from the puparium generally in 5 days.

"6. The fly of either sex takes its initial bite in 6 to 8 hours after emergence from the puparium. Flies of this species have been observed to feed experimentally on 17 species of vertebrates including man, reptile, bird, and rodent.

"7. It has been demonstrated that in feeding on live stock *Stomoxys* probes a wound with its labium from which nonbiting flies draw blood. Surra organisms have been demonstrated in the mouth parts and stomachs of house flies used in experiments in this connection.

"8. In considering the longevity of *Stomoxys calcitrans* it has been determined that a female can live a maximum of at least 72 days and a male a period of 94 days.

"9. The life cycle of *calcitrans* varies considerably according to the treatment the young forms receive. Under optimum conditions this is a period of 12 days, but under unfavorable surroundings in light and absence of moisture, the life cycle may be extended to 35 days."

G. C. L.

MESNIL (F.). *Maladies Infectieuses et Invertébrés Transmetteurs*.—*Bull. Inst. Pasteur.* 1913. Mar. 15. Vol. 11. No. 5. pp. 185-196; and Mar. 30. No. 6. pp. 233-244.

A very full and interesting resumé of the infectious maladies transmitted by invertebrate hosts. A zoological list of the invertebrates that convey disease is first given, then a review of the infectious maladies carried by insects or other invertebrates, and lastly an account of the invertebrates and the micro-organisms which they convey. Mechanical transmission, hereditary transmission and many other interesting points in connection with the subject are described. A bibliography of 61 papers dealing with the more recent work is given at the end of the paper.

G. C. L.

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**BOOK REVIEW.**

**MINETT (E. P.).** **Diagnosis of Bacteria and Blood Parasites.** 2nd Edition. Crown 8vo. viii. + 80 pp. 1913. London: Baillière, Tindall & Cox. [2s. 6d. net.]

A useful little work on the identification of bacteria, their staining reactions, etc. Special reactions—Wassermann's, Widal's, Calmette's, and the opsonic index—are also described. Owing to the great advances made by tropical medicine in the last decade the author has left it to be his duty to incorporate a short summary of the more common blood parasites. To make room for this, without materially increasing the size of the book, many duplicates of staining methods have been deleted.

G. C. L.

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## TROPICAL DISEASES BUREAU.

TROPICAL DISEASES  
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## KALA AZAR.

## EXPERIMENTAL.

BASILE (Carlo). *La Trasmissione sperimentale delle Leishmaniosi del Mediterraneo ai topi per mezzo delle pulci.* [The Transmission of Mediterranean Leishmaniasis to Rats by means of Fleas.] — *Atti della Reale Accad. d. Lincei. Rendiconti.* 1913. Apr. 6.

The author reminds his readers that he has shown that kala azar in the Mediterranean districts is transmitted amongst dogs and human beings by the human and dog fleas, and that in these the leishmania appear as flagellates which are morphologically distinct from the flagellates peculiar to these fleas. He has obtained a further confirmation of this view in the following manner.

On 25th and 26th January 1913, there were caught from a dog heavily infected with leishmania 200 fleas. These were fixed on wires by NÖLLER's method and kept at a temperature of 22° C. The day following their capture the fleas were made to feed upon a new-born dog in order that their faeces might be examined. Only three of the fleas showed flagellates of the type leishmania in their faeces. One was only slightly infected, the other two heavily. The two heavily infected fleas were dissected and an emulsion made in saline at 22° C. Part of this emulsion was injected intraperitoneally into two white rats, while the rest was made into films which showed abundance of flagellates. The marrow of the two rats was examined on February 26 by amputation of one hind limb and again on March 10 by removal of the other. Both these examinations were negative. On March 22 (56th day after injection) one rat was killed and in the hypertrophied spleen leishmania were discovered. This proves to the author that his views are correct and that the flagellates in the fleas were in reality leishmania.

[The reviewer has had some experience of NÖLLER's method (this *Bulletin*, Vol. 1, p. 367) and has found that the fixing and

examination of a single flea may occupy an hour or more. Basile appears to have fixed, fed, and collected the excreta of 200 fleas in two days.]

C. M. Wenyon.

SCORDO. A proposito di Alcuni Tentativi d'Infezione delle "Anopheles" con Succo Splenico di Malati di Leishmaniosi Interna. [Some attempts to infect Anopheles with splenic juice from Leishmaniasis patients.]—*Malaria e Malat. d. Paesi Caldi*. 1913. Mar. Vol. 4. No. 2. pp. 84-89. With 1 plate.

In a former paper (this *Bulletin*, Vol. 1, p. 633), the author described his attempts to infect Anopheline mosquitoes by feeding them on cultures of leishmania. He has now made similar experiments with the leishmania obtained directly from the spleen of a case of infantile kala azar. The blood which was rich in leishmania was treated in three ways. Firstly, some of it was placed on the surface of a disc of flesh over which was inverted a glass containing mosquitoes. Secondly, a piece of flesh was made to soak up some of the blood after which it was suspended in gauze in a glass with mosquitoes. Thirdly, some of the blood was injected into the superficial layers of the skin of a rabbit. The mosquitoes were allowed to remain for a quarter of an hour over the pieces of flesh when they were placed to feed upon the rabbit at the area of injection of the leishmania. About sixty mosquitoes were employed in the experiment and of these forty were eventually dissected after an interval of 24-48 hours or longer. In some of the mosquitoes no evidence was obtained of their having fed as there was no blood in their intestines. In others unaltered red blood corpuscles were present but never any leishmania. The salivary glands also gave negative results. As a result of these experiments therefore the author not only failed to obtain any evidence of a development of the leishmania in the mosquitoes but was unable to prove that the leishmania had been ingested.

In two smears made from the mosquitoes the author encountered an organism which he thinks is of protozoal nature and which is readily mistaken for leishmania on account of its similarity in size. The organism is pear-shaped with one extremity rounded and the other pointed, and is frequently grouped in clusters of six to eight enclosed in a kind of capsule which takes a pink tint in the stained films. Free forms are also present. There is a red staining nucleus at the centre and usually a more deeply staining granule at the pointed extremity. The author does not commit himself as to whether these correspond to the two nuclei of a leishmania. Division forms were not numerous though some with two nuclei were seen. The parasites were very numerous, as many as a hundred occurring in a single field of the microscope. The mosquito in which this organism was found was at the third day of experiment but there was no evidence, in the shape of altered or unaltered red blood corpuscles, that it had taken up any blood. This fact together with the numbers present has convinced the author that the organism is not a developmental

form of the leishmania. A coloured plate with thirteen figures illustrates this parasite, no previous reference to which can be found by the author.

C. M. W.

SCORDO (Francesco). *Die Leukocyten des Meerschweinchens und des Kaninchens in Kontakt mit den Flagellatenformen der Leishmania donovani in vitro und im Körper der Tiere.*—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. May 3. Vol. 69. No. 1-2. pp. 85-89. With 1 plate.

This is a German translation of the author's paper which has already been reviewed (see this *Bulletin*, Vol. 1, p. 364).

C. M. W.

#### EPIDEMIOLOGICAL AND CLINICAL.

CARONIA (G.). *Weiterer Beitrag zur Leishmania Anemie.* [Further Contribution to the Study of Leishmaniasis.] — *Arch. f. Kinderheilkunde.* 1913. Feb. 20. Vol. 59. No. 5-6. pp. 321-347.

This paper contains an account of 37 cases of infantile kala azar which have been seen in the clinic for children's diseases at Palermo University since April 1911. Twenty pages are devoted to details of the history of the cases while the rest of the paper deals with general matters arising therefrom. As in former accounts of cases from Palermo it is again shown that the disease attacks especially the children of the labouring classes who live in small rooms together with all kinds of domestic animals. Infantile kala azar most usually commences in winter or early spring.

As regards the condition of the blood it was noted that in about one half of the cases there was a leucopenia while in the other half the number of white blood corpuscles was hardly changed from the normal.

There were only two cases of recovery out of the total of 37. Fifteen of the cases were examined with the object of testing the blood for the presence or absence of antibodies. In only one of these was there a partial fixation of the complement while in another case in which a recovery took place there was complete fixation. This case has been the subject of a paper by DI CRISTINA and CARONIA (see this *Bulletin*, Vol. 1, p. 7). A study of the cases shows that they arise in several small centres scattered round Palermo. Only two cases arose in Palermo itself. The relation of dogs to the disease in children has been the subject of a special enquiry but only in one case was an infected dog found, in a centre to the north of Palermo; later in conjunction with BASILE the author discovered another in Carini, an endemic centre 25 kilometres from Palermo.

As a result of his observations the author believes that the disease is not always acquired in the same way and that there exists in all probability a variety of mechanisms by which kala azar is transmitted.

C. M. W.

SPAGNOLIO (G.). *Nota Clinica su Alcuni recenti Casi di Leishmaniosi Interna (Kala-Azar)*. [Some Recent Cases of Kala Azar.]—*Malaria e Malat. dei Paesi Caldi*. 1913. Mar. Vol. 4. No. 2. pp. 80-82.—*Riforma Medica*. 1913. May 17. Vol. 29. No. 20. pp. 536-538.

In a paper published in November 1912 the author drew attention to fifteen cases of kala azar (see this *Bulletin*, Vol. 1, p. 361). He now gives a description of a further eleven cases from the district of Bordonaro in Sicily, one of which is apparently recovering. All the cases, which are typical, are in young children the eldest of whom is three and a half years of age. In only two had the children associated with dogs in their homes and in one the author knew the dog was perfectly healthy. He calls attention to the following three facts in connection with this series.—

1. In the houses from which the cases came lived other children of various ages who were in the best of health.

2. In two cases where children had previously died of the disease in the same house a considerable interval had elapsed between the death of one and the falling ill of the other.

3. No evidence could be obtained associating the occurrence of the disease in the children with the death of any dog in the vicinity of the house.

The author believes that these three factors render the canine origin of the human cases very doubtful and more so the idea that fleas are responsible for the transmission of the malady.

C. M. W.

SALVATORE (Domenico). *Un Caso di Kala Azar a Derna*.—*Malaria e Malat. d. Paesi Caldi*. 1913. Mar. Vol. 4. No. 2. pp. 73-76.

The case described is that of a soldier who contracted the disease either at Derna or some other Italian town. The usual features were presented by the case which terminated fatally owing to violent haematemesis. Leishmania were discovered post mortem, thus confirming the diagnosis made clinically before death. The author points out that this, being a case in an adult, still further supports the view of GABBI that no real difference exists between the Indian and Mediterranean diseases, for both of which there is the single parasite *Leishmania donovani*.

C. M. W.

CIMINATA (Antonino). *Sul Reperto Parassitario nelle Anemie da Leishmania*. [The Finding of Parasites in Leishmaniasis.]—*Rivista Ospedaliera*. 1913. Feb. 28. Vol. 3. No. 4. pp. 156-159.

The author describes two cases of infantile kala azar which were examined post mortem. Smears made from the spleen and bone marrow and stained by Romanowsky stains did not reveal any leishmania though in an examination of sections of the various organs leishmania were seen in fair numbers in the

endothelial cells of the capillaries. Reference is made to a similar case seen by DI CRISTINA; leishmania were discovered in numbers in material drawn from the spleen during life though smears made from the spleen soon after death failed to show any; in sections however typical cells containing parasites were seen. This possible absence of leishmania in smears renders the diagnosis of kala azar very difficult. According to DI CRISTINA the disappearance of the parasites from the spleen soon after death, though they may have been present in large numbers before, is due to the presence in the blood of some lytic substance which destroys the majority of the leishmania.

[A similar case of the disappearance of leishmania soon after death was recorded by LIGNOS (see this *Bulletin*, Vol. 1, p. 4), and the case described by ARCHIBALD (see below) in which parasites could not be discovered in smears is of interest in this connection].

C. M. W.

GOBETTI (Girolamo). **Su di una Forma rara di Splenomegalia. Contributo Anatomico-Patologico.** [A rare Form of Splenomegaly.] —*Malaria e Malt. d. Paesi Caldi.* 1913. Mar. Vol. 4. No. 2. pp. 117-122.

The case described is that of a youth aged seventeen from Monteleone in Calabria, who suffered from fever with enlargement of the spleen. The disease terminated fatally and the author made a detailed examination of the organs, an account (five pages) of which is given. Though a careful search was made no leishmania could be discovered in any of the organs.

C. M. W.

ARCHIBALD (R. G.). **An interesting Case of Kala Azar.**—*Jl. R. Army Med. Corps.* 1913. May. Vol. 20. No. 5. pp. 512-521. With 1 plate.

The case is that of a young Arab, aged twenty, who fell ill in Khartoum with a disease which ultimately turned out to be kala azar contracted at Singa on the Blue Nile. A most interesting feature of the case was the failure to find leishmania in smears though puncture of the liver was performed four times and of the spleen three times. A diagnosis of kala azar was only obtained by the finding of leishmania in the liver of a monkey (*Cercopithecus sebaeus*) which had been inoculated intraperitoneally twelve weeks before, with material obtained from the case by liver puncture. Culture tubes of N.N.N. medium inoculated from the liver and spleen puncture material and incubated at 24° C. also failed to develop flagellates.

It was noted on the several occasions that liver and spleen puncture were performed that the material so obtained was of a granular nature, and in the stained smears peculiar "coccal bodies" were found of varying shades of blue and red and enclosed as a rule in cells. They resembled to all intents and

purposes large cocci more or less uniform in size massed together in the cells, which were devoid of nuclei and had a blue and often vacuolated protoplasm. In the cultures on N.N.N. medium, though no flagellates developed, the "coccal bodies" were present even after ten days though they showed no sign of development.

As regards the nature of the bodies the author is somewhat guarded in his views but he says they gave one the impression of being coccal organisms of a specially large type. He admits that the nuclei of some of the cells in the smears showed granular changes. Similar bodies had been previously found by the author in smears from another case of kala azar from the same district. He draws attention to the fact that CARTER and THOMPSON and BALFOUR record the presence of large coccal bodies in oriental sores of the non-ulcerating type, and SEIDELIN has recently described a coccus in four cases of dermal leishmaniasis from Yucatan.

As regards the examinations of the peripheral blood in this case, there was a great increase in the large lymphocytes, a change which in the author's experience is a very constant feature of kala azar in the Sudan. There was also an increase in the eosinophile cells which appeared to be unconnected in any way with skin lesions or worm infections of the gut. Leishmania were never seen in the blood films.

Though the case was undoubtedly one of kala azar as demonstrated by the monkey inoculation, the patient recovered without specific treatment.

C. M. W.

CRESPIN. *Leishmaniose et Paludism chronique infantile.*—*Caducée*. 1913. Apr. 5. Vol. 13. No. 7. p. 89.

The author gives a table illustrating the differences between the two easily confused diseases chronic infantile malaria and kala azar. In malaria there is the early and almost simultaneous appearance of the cardinal symptoms, fever, gastro-intestinal trouble, enlargement of the liver and spleen, oedema, and anaemia with an earthy colour of the skin. In kala azar the same symptoms appear, but slowly and successively—first the fever, then the intestinal symptoms, and finally the others.

In malaria the large liver and spleen are painful and tender and there are referred pains along the course of the phrenic nerve, while in kala azar these organs are neither painful nor tender and there are no referred pains. In malaria there is more usually constipation rather than diarrhoea while in kala azar the reverse is the case. In malaria oedema of the face and lower extremities is the rule, while in kala azar it is limited usually to the lower extremities. With malaria recovery is the rule after specific treatment, while in kala azar there is no specific treatment and the disease is incurable.

C. M. W.

LOMBARDO (Giacomo). **Contributo allo Studio delle Alterazioni Anatomiche dell' Anemia da Leishmania.** [Anatomical changes found in Leishmaniasis.]—*Pathologica*. 1913. May 15. Vol. 5. No. 109. pp. 292-296.

This paper contains a detailed account of the post mortem findings in a case of infantile kala azar from Catania.

C. M. W.

#### TREATMENT.

CARONIA (G.). **Sulla Guaribilità dell' Anemia da Leishmania (a proposito di 8 casi di Guarigione osservati a Palermo).** [Eight Cases of Recovery from Leishmaniasis.]—*Malaria e Malat. d. Paesi Caldi*. 1913. Mar. Vol. 4. No. 2. pp. 90-96.

Though it was first held by JEMMA that infantile kala azar always terminated fatally, JEMMA, BASILE, NICOLLE and others gradually realised that a mild form of the disease existed and complete recovery was possible. JEMMA described a case of recovery in a child five years old from Palermo and NICOLLE another in a child of two from Tunis. Two other cases were recorded by SPAGNOLIO in Messina and LIGNOS in Athens, while JEMMA has had three further cases of recovery in Palermo. In the present paper the author gives an account of the cases of recovery met with at the clinic of JEMMA at Palermo. Seventy-eight cases of infantile kala azar have been seen. Of these fifty have died, eight have recovered, while twelve are still living and eight have been lost sight of. Neglecting those living and those that have disappeared the percentage of recoveries is about fourteen. Outside Palermo other cases of recovery have been described by NICOLLE, SPAGNOLIO, LIGNOS, PETRONE, and CARTOPHYLLIS and SOTIRIADES, making a total of fourteen recorded recoveries from infantile kala azar in the Mediterranean districts.

The author describes in detail the eight cases from Palermo. The ages varied from 21 months to five years and in seven cases leishmania had been discovered, usually in large numbers in smears from the spleen. In the single case in which leishmania had not been seen microscopically a dog inoculated with spleen pulp contracted the disease. As regards treatment various remedies have been used, but the author believes that in each case the cure resulted from natural processes though he is of opinion that arsenic, in whatever form administered, has a beneficial action.

C. M. W.

ROUX (F.). **Arsenic in the Treatment of Kala Azar.**—*Indian Med. Gaz.* 1913. Apr. Vol. 48. No. 4. pp. 132-133.

The author has treated cases of kala azar by means of Ramaline, a French composition containing arsenic in the form of a compound of mineral and organic salts. This compound offers the great advantage over pure arsenic of being supported without ill result by the patient. It is administered



in the form of a pill containing 1 milligramme of arsenical salts and the patient is given eight pills a day. So far five cases have been treated with favourable results.

C. M. W.

#### CANINE KALA AZAR.

**BASILE (Carlo).** *La Trasmissione sperimentale della Leishmaniosi naturale del cane ai topi conigli e cavia.* [The Transmission of Canine Leishmaniasis to Rats, Rabbits, and Guinea-pigs.] — *Atti della Reale Accad. dei Lincei, Rendiconti.* 1913. Mar. 16.

Two rats and two rabbits were injected intraperitoneally with an emulsion of the organs of a dog infected with leishmania. One rat was killed thirty days later and the slightly enlarged spleen was found to be infected with leishmania. The second rat is still under observation. The two rabbits killed on the thirtieth day gave a negative result, while similarly five young guinea-pigs were unsuccessfully inoculated intraperitoneally from the organs of an infected dog.

C. M. W.

**BASILE (Carlo).** *Sulla Leishmaniosi nel cane e sull'Esistenza di Leishmania nel midollo spinale di cani naturalmente infetti.* [Leishmaniasis in the Dog. Leishmania found in the Spinal Marrow of Naturally Infected Dogs.] — *Atti della Reale Accad. de Lincei, Rendiconti.* 1913. Apr. 20.

Canine kala azar is characterised clinically by fever, alopecia, wasting, and paraplegia of the hind limbs and can develop in either an acute or a chronic form. The acute type of disease attacks dogs of about five months of age, while the more chronic form is seen in older dogs, which frequently recover naturally and may be apparently healthy, though harbouring the parasite for considerable periods of time. In the chronic form, when a fatal termination is threatening, the nervous symptoms, involving especially the hind limbs, become more marked so that it would appear that there is some definite involvement of the nervous system. This view the author has confirmed by the finding of leishmania in smears of the spinal cord of two dogs naturally infected with kala azar. One of these dogs was presented to the author by the father of a child who was at the time suffering from kala azar. The dog was subjected to the operation of splenectomy for purposes of experiment but it died during the operation. The spleen was of normal dimensions, a fact which suggests to the author that forms of anaemia in children who have no enlargement of the spleen may still be kala azar.

LA CAVA was the first to demonstrate leishmania in the cerebro-spinal fluid of a case of human kala azar and the author now draws attention to this condition in dogs and points out that the nervous symptoms of both kala azar and trypanosomiasis are due to a similar cause.

C. M. W.

## TROPICAL SORE.

WENYON (C. M.). **A Further Note on a Case of Dermal Leishmaniasis from S. America, with the Results of Inoculation Experiments.**—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. Part 2. pp. 117-119.

This case has already been described (see this *Bulletin*, Vol. 1, p. 13). The present note deals with the subsequent history after treatment was commenced, the result of the culture of the leishmania obtained from it, and some inoculation experiments on animals.

It is recorded that the patient was treated in two ways, firstly by killed cultures of the leishmania injected subcutaneously and secondly by the application of an ointment made of equal parts of methylene-blue, lanoline and vaseline. This ointment was applied on a piece of lint the size of the sore both morning and evening. Healing quickly took place, apparently under the influence of the ointment.

Pure cultures were obtained from the sores and with these some inoculation experiments were carried out:—

"1. With the first culture obtained (Nicolle's culture virus) a human being was inoculated on the skin of the forearm in two places—one by intra-cutaneous injection with a hypodermic needle, and the other by scarification. A year has now elapsed since the inoculation and no infection has taken place.

"2. A young rabbit was inoculated intravenously on two occasions with rich cultures of the leishmania, but no infection took place.

"3. A young rabbit was inoculated intravenously on one occasion with rich culture, but did not become infected.

"4. Attempts were also made to infect mice by intraperitoneal injection of rich cultures, but without success."

With the virus obtained from the sore a dog and baboon were successfully inoculated but a small puppy injected intravenously failed to acquire the infection. Two mice inoculated intraperitoneally did not become infected. A cat inoculated in each ear developed small pinhead nodules after two months at the sites of inoculation. These have increased slightly in size and now, five months after inoculation, are still present, and leishmania have been discovered in them first by the culture method on N.N.N. medium and later by microscopic examination of smears from the sore.

Compared with a culture of *Leishmania tropica*, obtained from the oriental sore of Aleppo, the South American leishmania grew much more readily, though morphologically no constant differences could be made out between the two types of parasite.

G. C. Low.

BATES (L. B.). **Leishmaniasis (Oriental Sore) of the Nasal Mucosa.**—*Jl. Amer. Med. Assoc.* 1913. Mar. 22. Vol. 60. No. 12. p. 898.

The case here described is the fifth of oriental sore encountered in Ancon Hospital. The patient, a farmer aged 35,

had suffered from the disease for three years. There was ulceration of the helix of each ear, of the free margins of the alae nasi, and the extensor surface of each elbow, a partially healed ulcer over the sternum, and a nodule on the skin. Smears from the ears and alae nasi showed leishmania, as also did smears made from the nasal septum on each side. There was thus definite involvement of the mucous membrane of the nose by the leishmania. This is the first case with this complication noted from Panama and suggests that the dermal leishmaniasis of Central America is identical with the disease of South America where involvement of the mucosae has been frequently described.

C. M. W.

ESCOMEL. **Première Déconverte de *Leishmania tropica* flagellée dans le Corps Humain.** [Correspondence.]—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 237-238.

The author writes that on September 6, 1911, he communicated to the *Sociedad medica d'Arequipa* his discovery of flagellated forms of leishmania in skin lesions. On two later occasions similar flagellates were discovered by him in other cases. He believes that this leishmaniasis is different from espundia and that the mucous membranes do not become involved. Accordingly he suggests that the organism should be distinguished under the name of *Leishmania americana* var. *flagellata*. A similar discovery of flagellated forms of leishmania was reported by LA CAVA (this *Bulletin*, Vol. 1, p. 370). LAVERAN, who presented this note to the *Société de Pathologie Exotique*, points out that it is premature to create a new species of leishmania on the occasional presence of flagellated forms in the lesions.

C. M. W.

KEELAN (R. S.). **A Case of Bagdad Sores showing an apparently long Incubation period.** [Mirror.]—*Indian Med. Gaz.* 1913. Apr. Vol. 48. No. 4. p. 146.

The note refers to a native aged 55 who had been all his lifetime in Lucknow. He made one pilgrimage to Bagdad a year and a half after which there developed sores on his hand and legs. These increased in size and were identified as oriental sore by the finding of leishmania.

C. M. W.

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## SLEEPING SICKNESS.

KLEINE (F. K.) & ECKARD (B.). Zur Epidemiologie der Schlafkrankheit. [On the Epidemiology of Sleeping Sickness.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. May. Vol. 17. No. 10. pp. 325-328.

In those regions with which the authors are acquainted sleeping sickness exhibits a tendency to spread but slowly. An isolated focus of the disease was found in Maisome, an island in Lake Victoria. At the landing place there lived four infected persons who judging from their symptoms had been ill for two or three years. These people had never left the island and must have been infected in the place. Infected sailors of visiting boats had been the reservoir from which the fly had derived its infection. Although the island swarmed with *Glossina palpalis* the infection had not spread, and the remaining 500 inhabitants were healthy. A similar state of affairs was found in south Tanganyika by STOLOWSKY and also by KINGHORN and MONTGOMERY (*Sleeping Sickness Bulletin*, Vol. 2, p. 384). The authors remark that the slowness with which the infection spreads in such regions is very astonishing. HORN (*Sleeping Sickness Bulletin*, Vol. 2, p. 224) and KINGHORN (*Sleeping Sickness Bulletin*, Vol. 2, p. 257) have explained the slow spread of the disease on the Gold Coast by the assumption that the population have acquired a certain degree of immunity. If, however, immunity occurred as a result of infection the number of healthy parasite carriers would be great, as is the case in malaria or in Texas fever or piroplasmiasis of dogs. Experience in Uganda and German East Africa has shown, however, that the great proportion of the infected persons died in the course of a few years. A natural or an acquired immunity has not been observed.

Experiments were undertaken to ascertain whether the tsetse flies developed any immunity to the parasites. It was thought that possibly young tsetse hatched from the pupae of flies which had been fed on infected blood might have acquired a certain degree of immunity, which would inhibit the development of the trypanosomes in them when they were subsequently fed on infected blood. Since the antibodies formed by the mammalian host during trypanosome infection are not strongly specific, the possibility is not excluded that *Glossina* through taking up one species of trypanosome might become non-receptive for another. In order to obtain information on this subject a large number of female *Glossina* were fed alternatively on sleeping sickness monkeys and goats infected with *T. congolense*. By this means the formation of antibodies against trypanosomes should be stimulated in the body of the mother flies. After the course of three weeks the pupae were collected as they were deposited and the flies—434 in number—which were subsequently hatched, fed on sleeping sickness monkeys. On the eighteenth day the surviving 402 flies were killed and examined. Eleven, or 2·7 per cent., were found to be infected. In another series of 90 *Glossina* the experiment was continued for a month and then the flies were put to feed in batches on three different monkeys; all

three became infected, showing that of the 90 at least three were infective. This percentage corresponds to figures obtained under similar conditions with young *Glossina* whose ancestors had not been previously fed on infected animals in the manner described. There is hence no reason to believe that there are developed in the bodies of flies, by the daily taking up of trypanosomes, antibodies which are transmitted to the next generation.

The slow spread of sleeping sickness can therefore be no more explained by an immunity of *Glossina* than by an immunity of man. The critical point must be sought in the nature of the trypanosome itself. It is impossible to infect *Glossina* with certain strains. Moreover, it has been shown by various workers that climatic conditions exert an important influence on the development of the parasites in the tsetse flies.

[The work of KINGHORN and YORKE in the Luangwa Valley (*Sleeping Sickness Bulletin*, Vol. 4, p. 315) showed that the proportion of infective (with *T. rhodesiense*) to non-infective wild *Glossina morsitans* was about 1 to 500, and that whilst about 16 per cent. of the big game were infected with this parasite yet only a relatively very small number of cases of sleeping sickness exist. Thus in a district where there is a sufficient number of infective fly to infect 16 per cent. of the wild fauna the disease appears to be spreading but slowly amongst the human population. Such data as this seem to indicate that perhaps man is for some reason less easily infected than the lower animals. It must be remembered that amongst the wild fauna themselves there appears to be a great difference in susceptibility to infection: waterbuck, for example, were found by KINGHORN and YORKE to be frequently infected with *T. rhodesiense*, whilst zebra were never found to harbour the parasites.]

W. Yorke.

SCHUBERG (A.) & BÖING (W.). Ueber den Weg der Infektion bei Trypanosomen- und Spirochätenerkrankungen. [The Track of Infection in Trypanosome and Spirochaete Diseases.]—*Deut. Med. Wochenschr.* 1913. May 8. Vol. 39. No. 19. p. 877-879.

Notwithstanding the enormous literature dealing with trypanomiasis little or nothing is to be found concerning the manner in which the parasites actually invade the vertebrate organism. Experiments were devised to obtain information on this subject.

In the earlier series a large number of punctures, of as little depth as possible, were made close together in a small area of skin. By this means the site of the punctures was easily found when sections of the skin were subsequently made. The punctures were made with the finest entomological needles. Infected blood was then dropped on the punctured area and allowed to remain there for some time. The behaviour of the ingoing trypanosomes was studied by examination of sections of the affected region. As by this method the number of invading trypanosomes was relatively very small the following technique was adopted in later experiments. Heavily infected blood, diluted with an equal volume

of 5 per cent. sodium citrate solution, was injected intracutaneously by means of a syringe. As far as possible the injections were made immediately under the epidermis or even into the corium, but not into the subcutaneous connective tissue. Owing to the thinness of the skin of rats and mice, which were the animals used, this procedure was quite easy. In the skin, and likewise in the subcutaneous tissue if this were pierced, distinct vesicles were formed which remained visible externally for several hours. This manner of introduction of the virus resembles closely that of the biting fly, with the advantage that many more parasites were injected and thus their behaviour could be more readily observed. In the earlier experiments the injected vesicles were situated on the abdomen, but later exclusively on the thigh because it was found desirable to examine the neighbouring lymphatic glands. Sections of the tissues involved were stained by Schuberg's modification of the Giemsa method. Experiments were carried out with *T. lewisi* and *T. brucei*.

By killing the various animals at different periods after the injection of infected blood and examining the tissues involved the authors were enabled to follow the path of the invading parasites.

The trypanosomes escape very quickly from the blood vesicle into the lymph spaces of the corium, and thence migrate through the subcutaneous connective tissue into the neighbouring lymphatic glands. In these sites they increase enormously in numbers and are to be observed in the glands of the same side of the body in large numbers before they can be found in the peripheral blood. It follows that the trypanosomes investigated (*T. lewisi* and *T. brucei*)—and probably all others—must be regarded not merely as blood parasites, but in not less degree as parasites of the lymphatic system.

The spirochaetes of relapsing fever and the fowl spirochaetes behave in a similar manner. They quickly escape from the injected blood and migrate into the surrounding connective tissue of the skin.

In a postscript Schuberg refers to the experiments of UHLENHUTH and EMMERICH (this *Bulletin*, Vol. 1, p. 677) which demonstrated that *T. gambiense* and *T. equiperdum*, when injected directly into the testes of rabbits, multiply rapidly in these organs; and further that sometimes even when these parasites were injected intravenously, a greater increase of the trypanosomes was observable in the testes than in the blood stream and other organs. In view of the observations recorded in the present paper UHLENHUTH expressed the opinion that the explanation might be that the testes contain numerous lymphatics.

W. Y.

HECKENROTH. *Tournée médicale effectuée sur le Congo et l'Oubangui.*—*Ann. d'Hyg. et Med. Coloniales*, 1913. Vol. 16. No. 1. pp. 104-144. With 3 maps.

The objects of this expedition, which started on September 25, 1911, were firstly to determine the therapeutic value of arsenophenylglycin in trypanosomiasis and secondly to obtain more precise information regarding the distribution of the disease.

The results obtained by treating sleeping sickness patients with arsenophenylglycin are published in another paper (this *Bulletin* Vol. 2. p. 35).

*Distribution of human trypanosomiasis on the middle Congo and Oubangui.*—The map published by the Commission to the French Congo in 1909, which is the only one we possess, required to be completed and revised. The number of natives examined was 9,359; direct examination of the blood and gland juice in conjunction with clinical examination was considered sufficient. The author studied successively the four districts of *Bangala*, *Bondjo*, *M'Baca* and *Banziri-Bouraca-Sango*.

*Bangala* extends along the French bank of the Congo from the northern extremity of the "Couloir" to the village of Liranga. Since 1907, when LEBOEUF visited the country, the population has considerably diminished and in certain parts has completely disappeared. In four years the population of the village of Loukoléla has been reduced to one-half. The villages in the Bangala district can be divided into four groups, viz., Bonga, Loukoléla, Iréhou and Liranga. The percentages of cases of sleeping sickness found at these were 3·87, 10·3, 5·69 and 9·93, respectively. It is interesting to note that in 1907 LEBOEUF gave the following figures—Loukoléla 13·3 per cent., Iréhou 16·04 per cent. and Liranga 15·5 per cent.

*Bondjo* extends on the Oubangui from the village of Boubangui to the great agglomeration of Ikoumba, a distance of about 350 kilometres. The results of examination of the population in the various groups of villages in this district are as follows:—Impfondo 5·24 per cent. infected, Doungou 1·3 per cent. (in one village, Boumba, 7 cases were found out of 39 persons examined), Bétou 7·9 per cent.

*M'Baca.*—The population of this district has been decimated by an exceedingly severe epidemic. It commences on the other side of Ikoumba and ends north of Bangui. LEBOEUF visited one of the villages, Bimbo, of this district in 1907. He considered that the first case of the disease did not occur before 1906; nevertheless he wrote, "The disease is about to make real progress at Bimbo and before long a heavy mortality is to be expected." This prediction has soon proved true and the author found that 39 per cent. of the 403 persons examined were infected. The other groups of villages examined in this district were Mongoumba where 20·5 per cent. and Yacoli where 27·2 per cent. of the population were infected.

*Banziri-Bouraca-Sango.*—This district reaches on the banks of the Oubangui from Bangui to Mobaye. In the Mobaye group of villages 9 per cent. of the population were infected, in the villages between Mobaye and Konango 15·7 per cent., in the Konango group 14·4 per cent., in the Bessou group 8·5 per cent., in the Kémo group 12·5 per cent., and in those between Possel and Bangui 19·7 per cent.

The above figures only apply to villages which are on the banks of the Congo and Oubangui, but in addition persons coming from the interior were met and examined at several of the river villages. Time did not permit the author to visit many villages

lying some hours' march away from the river. Of 1,088 persons examined who came from such villages 5 per cent. were infected. There is little infection amongst the population who live constantly at a distance from the Oubangui. Those who, on the contrary, frequently visit the river as porters and labourers are fairly heavily infected. The author points out the necessity for supervision of the movements of natives, to protect regions still free from infection. The question is of the utmost gravity, since it is well known with what rapidity countries are devastated where trypanosomiasis assumes its epidemic form. Half of the population of the river has been annihilated in less than ten years.

*Prophylaxis of human trypanosomiasis on the middle Congo and Oubangui.*—Human trypanosomiasis has spread along the Congo and Oubangui to install itself at length on their tributaries, which with the commercial development and administrative occupation of the country have gradually become important highways; e.g. the M'poko, Lobaye, Tomi and Kotto.

As a rule the percentage of trypanosomiasis is greater in the factories and on the stations than in the neighbouring villages, but occasionally, especially during epidemics, it may be higher in the villages. This anomaly is perhaps only apparent, as during epidemics the natives who are in the employ of the European are better fed, lodged and clothed than their fellows in the villages. It is probably such factors that explain why Europeans are rarely infected although they may spend considerable periods in villages where 25-30 per cent. of the population are infected.

*Glossina palpalis* was found everywhere along the middle Congo and the Oubangui. Only two specimens of *Glossina fusca* were taken. It was found that the distribution of tsetse did not correspond with that of sleeping sickness, or more precisely, the "coefficient tsétsé" is far from explaining the mortality from sleeping sickness in all districts. For example at Bonga, Loukoléla and Irébou there are innumerable tsetse which are found even in the village clearings and make travelling by canoe a veritable torment. Notwithstanding this very high "coefficient tsétsé"—higher than in any other portion of the Congo or Oubangui visited by the author—very few cases of sleeping sickness were found. On the contrary in Mobaye and Kémo, where 15-30 per cent. of the population are infected, tsetse is rare on the French bank during this period of the year (December and January). The natives assert, however, that the fly is numerous on the Belgian bank. Under these circumstances the question arises whether any other biting fly is concerned in the spread of the disease. In the course of his tour the author carefully noted the biting flies found in each village and district visited by him. Mosquitoes, Stomoxys, Tabanidae, Simuliidae and the larvae of *Auchmeromyia luteola* were encountered in various villages. No definite relationship could be established, however, between any of these and trypanosomiasis. The question of the transmission of sleeping sickness in countries where the tsetse does not exist can be solved only by a prolonged series of transmission experiments.



In a table details are given of the number of cases found at the various villages in each of the four districts. In the Bangala country 1,012 persons were examined and 58 cases found, in Bonjo there were 170 cases out of 2,373, in M'Baca 569 out of 2,022 and in the Banziri-Bouraca-Sango group 437 out of 3,372. The total population was estimated at 19,000 and the number of cases of sleeping sickness 2,550. These figures show the necessity for measures which will tend to protect the country against the ravages of trypanosomiasis and to prevent its extension to new regions. In the bush only recommendations as to general hygiene, and simple measures of prophylaxis such as clearing, isolation of the sick, and notification of fresh cases are practicable. Isolation of infected cases is practised, but only concerns those in the last stages of the disease. Of the 1,250 cases recognised by the author only 20 were kept aloof by the inhabitants. Isolation indeed appeared to be to the native less a matter of safeguard than of personal embarrassment, resulting from a relative or friend becoming too troublesome. The unfortunate isolated ones, badly nourished and finally totally abandoned, quickly succumb. This is probably why so little sleeping sickness in the third stage is seen.

From the point of view of prophylaxis the length of time which the untreated survive is of importance. The author is able to give information touching certain cases noted by LEBOTTE in 1907 which have remained untreated. At Loukoléla of 8 cases 2 have disappeared, 5 are dead and 1 is in good health on the Belgian side. At Irébon of 13 cases 4 have disappeared, 7 are dead and 2 are still living in the Belgian Congo. At Liranga of 14 cases 2 have disappeared, 1 died in 1907, 3 in 1908, 3 in 1909, and 2 in 1910. Of the remaining 3, 1 was in good health on the other bank and 2 were seen by Heckenroth and had apparently recovered.

W. Y.

**MITZMAIN (Maurice Bruin).** *The Rôle of Stomoxys calcitrans in the Transmission of Trypanosoma evansi.*—*Philippine Jl. Science.* Sect. B. (*Philippine Jl. Trop. Med.*) 1912. Dec. Vol. 7. No. 6. pp. 475-518. With 5 plates.

This paper records a considerable number of precise experiments devised with the object of ascertaining whether *Stomoxys calcitrans* can transmit *T. evansi*. All attempts to demonstrate mechanical transmission were negative; nor was success met with in efforts to effect transmission by means of interrupted feeding. The only positive result obtained was in one of three experiments to ascertain whether mechanical transmission could be effected by successive interrupted feeds.

Further experiments were conducted to see if there was a cyclical development of *T. evansi* in the fly. Both wild and laboratory bred *Stomoxys* were used in these experiments. In the first experiment 14 wild flies were used. They were allowed to feed once on a heavily infected guineapig and subsequently were fed daily on healthy animals. The experiment was continued until the 18th day, when the sole remaining fly was too

enfeebled to feed. It was inoculated subcutaneously into a healthy guineapig. Neither this nor any of the healthy animals upon which the flies were fed became infected. Three other experiments in which 190, 60 and 90 laboratory bred *Stomoxys* were employed also gave negative results. In the first two of these the flies were fed on two consecutive days on infected animals and in the third on three consecutive days.

An account is given of some interesting observations on the relation of non-biting flies to *Stomoxys* in contaminative infections. An abnormal percentage of non-biting flies was generally found on collecting insects from domestic animals, and the majority of them were found on examination to be engorged with mammalian blood. A quiet bullock was selected for observation; some 150-200 flies, mostly muscids, were seen to collect on him. In a short time the author's attention was attracted to the peculiar grouping of the ecto-parasites: groups of 2 to 4 and 5 prevailed. On closer inspection the group was found invariably to consist of more than one species, a *Stomoxys* usually providing the central figure. Where this species was lacking it was found that the group fed from a common area with the heads of the individuals in close contact; the food was found to be a droplet of freshly exuding blood. It was noticed that even other blood-sucking flies found on cattle often take advantage of the action of the more powerful proboscis of the *Stomoxys*. *Lyperosia* was found to await its turn with the non-biting flies.

Experiments were conducted to determine the relationship of the common house fly, *Musca domestica*, to *Stomoxys* as a harbourer and carrier of trypanosomes. Numerous trypanosomes were found in the abdominal contents, 3 hours after the flies had been fed on the abraded tail of an infected monkey, and inoculations of the abdominal contents into guineapigs resulted in infection. In another experiment 30 laboratory bred *Stomoxys* were fed on a healthy monkey. Immediately they had fed 50 *Musca* which had fed on a fresh wound on a surra animal were substituted for the *Stomoxys*. Full opportunity was thus given them to carry infected material on labella and pulvillus into the wounds presented. The results of this and three similar experiments were negative.

Details are given of the technique employed in keeping *Stomoxys* for experimental work. The greatest difficulty was encountered in attempting to keep flies, in either small or great numbers, in a common enclosure.

*Screened stable.*—Here the difficulties presented are summed up in the presence of natural enemies. Particular reference is made to the common insectivorous lizard and the ubiquitous spider. Spraying with cresol was effective in dealing with these.

*Glass vessels.*—Large bottles and museum jars of three litres capacity were used. Thirty days was the longest time flies were kept alive in these containers. Untimely death resulted from mite infestation, cannibalism, and excess of moisture.

*The use of individual glass tubes.*—By this means the difficult problem of keeping the flies alive in captivity was most successfully solved. Ninety-four days was found to be the maximum

life of the adult *Stomoxys*, kept individually under laboratory conditions. A test tube of 24 mm. bore plugged with cotton was found the most convenient; moisture requirements were regulated by means of pieces of filter paper which were changed at least every two or three days. The tubes were not changed oftener than twice a week. The flies when not feeding were kept in the dark at 20-26° C.

The following summary is given—

1. Only negative results were obtained in the attempts at direct mechanical transmission of surra with flies which were induced to bite healthy animals at intervals ranging from five minutes to three days after being permitted to complete the feeding upon infected animals. Thousands of *Stomoxys calcitrans* were employed in 29 experiments involving the use of 3 horses, 6 monkeys, and 22 guineapigs.

2. Twenty-seven experiments were performed in attempts to transmit surra by the interrupted method of feeding. All attempts proved negative where a single application of a varying number of flies was used, as many as 38 on a horse, and a maximum of 40 on a small guineapig. The intervals between feeding on infected and healthy animals averaged twenty-five to forty seconds in the two instances cited.

3. In 3 trials, interrupted feeding was employed in successive daily applications. In attempting to determine the minimum number of bites necessary to infect an animal, as high as 40 were followed by negative results. The only positive result obtained was produced from a succession of 206 interrupted bites in which the flies were transferred immediately from the infected to the clean animal. The flies were applied thirty-two hours during a period of six days.

4. The results of these experiments indicate that *Trypanosoma evansi* does not develop in the body of *Stomoxys calcitrans*. Ninety-four days was the longest period in which laboratory-bred flies were tested for a cyclical development, and sixty-seven days the maximum for wild flies.

5. Organisms of surra were not found in *Stomoxys calcitrans* beyond eighteen hours after feeding on an infected animal, and the limit for infection by inoculation was ascertained in these experiments to be six hours.

6. Pathogenic trypanosomes were found in the proboscis of the fly thirty seconds after feeding on infected blood. Within one minute and thirty seconds the organisms were not present in the mouth parts in a form capable of infecting by inoculation into guineapigs.

7. The wounds made by the labium of *Stomoxys* were not found to be a suitable channel for infection. Consequently, it is not likely that surra in domestic animals is produced through this avenue by external contamination; namely, faeces, mouth parts, and pulvilli of infected flies.

8. The intimate relation in the feeding habits of *Stomoxys* and of house flies has been pointed out. *Stomoxys* has been demonstrated to provide through its bites the infection of *Musca domestica* and other dung flies. These flies have been demonstrated to act as carriers, harbouring the surra organisms for several hours.

9. No evidence was obtained to indicate that *Tr. evansi* is hereditarily transmitted to the offspring of *S. calcitrans*. The larva of this fly fed on surra blood does not continue to harbour the trypanosome and the fly is "clean" upon reaching maturity.

10. It is demonstrated that the individual glass-tube method is the most suitable for applying flies in feeding on experimental animals and for keeping flies for long periods under laboratory conditions.

The photographs illustrate the methods of keeping flies for transmission experiments.

W. Y.

MITZMAIN (M. Bruin). Collected Notes on the Insect Transmission of Surra in Carabaos.—*Philippine Agricultural Review*. 1912. Dec. Vol. 5. No. 12. pp. 670-681.

The first portion of this paper deals with the dispersion of the lice of carabao—the Indian buffalo (*Bubalus bubalis*)—and their rôle in the transmission of *T. evansi*.

One thousand lice (*Haematopinus bituberculatus*), collected from a carabao whose blood contained numerous trypanosomes, were divided into five lots of 200 each. In one of these, set aside as a control, it was found that although the majority of the lice lived for two days none were alive after three days. The other four lots were fed on healthy carabaos 2, 10, 24 and 48 hours, respectively, after their removal from the infected host. Only that carabao became infected upon which the lice were fed which had been removed from the infected host but two hours previously.

It was found that the *Lyperosia* sp. fly which is common in the Philippines is responsible for the dispersal in nature of large numbers of the carabao lice. In a collection of 1,800 *Lyperosia* 620 lice were found attached to the flies: the lice were never found attached to *Stomoxys* or indeed to any other species than the *Lyperosia*.

It was observed that in carabao infected with surra the trypanosomes were frequently absent from the peripheral circulation for comparatively long periods. Susceptible animals were inoculated with the blood of infected carabao after these negative periods had lasted for from two to 26 days. As a result of these experiments it is concluded that during the negative stage of the disease the carabao is not a source of danger as an infective focus.

In an entomological study of a surra outbreak among carabaos *Lyperosia*, *Stomoxys*, *Tabanidae*, *Muscidae* (non-biting) were found in the various infected haciendas in the proportions given in the table.

Table II.—Fly distribution.—Percentage according to host.

—			Lyperosia.	Stomoxys.	Nonbiting Muscid.	Tabanids.
Cattle ...	...	...	70	10	20	3
Carabaos ...	...	...	95	3	2	specimens.
Horses ...	...	...	25	70	5	

The author points out that these observations are of but slight value as the study was of only two weeks' duration. He is of the opinion that the presence of carabao lice in unusual numbers, with due consideration to other insects, influences the incidence of surra.

An experiment was made to ascertain the relation of *Lyperosia* sp. to surra in carabaos. A fly-proof cage was so partitioned that two surra-infected carabaos and a healthy carabao could occupy adjoining stalls without bodily contact and still be

exposed to the bites of flies placed in the enclosure. In all, over 5,000 flies were employed in the experiment. The result was negative as the healthy animals did not become infected.

W. Y.

#### TREATMENT.

**KOLLE (W.), HARTOCH (O.), ROTHERMUNDT (M.), & SCHÜRMANN (W.).** Ueber neue Prinzipien und neue Präparate für die Therapie der Trypanosomeninfektionen. [On New Principles and New Preparations for the Treatment of Trypanosome Infections.]—*Deut. Med. Wochenschr.* 1913. May 1. Vol. 39. No. 18. pp. 825-828.

This paper records the results of experiments to ascertain the value of various preparations of antimony as therapeutic agents in trypanosomiasis.

In the earlier experiments mice infected with nagana were employed. The strain killed the mice regularly within five days. An emulsion of metallic antimony in oil, injected intramuscularly, was found to produce acute toxic symptoms and death, in doses of 5 mgm. per 10 gm. of body weight of the animal. It was determined that 1 mgm. of metallic antimony was sufficient to cure the infection with certainty. The trypanosomes disappeared from the circulation within 24 hours and did not return. Unfortunately, metallic antimony even in this dose proved poisonous. All the animals treated with metallic antimony and also with most other active preparations of antimony used by the authors died from chronic poisoning. The mice, however, died sterile; parasites were not found in the blood nor in any of the organs and subinoculated animals did not become infected. Antimony trioxide was an exception as it proved to be much less poisonous. Many other metallic preparations were used, but except in the largest doses had no therapeutic action on trypanosome infections.

Experiments were undertaken to ascertain whether the therapeutic activity of the various antimony compounds had any relation to their chemical constitution. It was found that pentavalent compounds were not toxic except in large doses, but also that they were not active therapeutically. Trivalent preparations proved to be exceedingly toxic and at the same time of great therapeutic activity. It was established that for antimony compounds—soluble or insoluble, organic or inorganic—to be active, the antimony must be in the trivalent form.

“Trixidin” (30 per cent. emulsion of antimony trioxide in oil) was found to have an extraordinary valuable therapeutic effect. This compound only killed mice in doses of 100 mgm. per 10 gm. body weight, and is thus practically non-toxic. No symptoms of chronic poisoning were observed after its administration. Intramuscular injection of 1 mgm. per 10 gm. of body weight cured mice with certainty.

As a result of the first part of their investigations the authors state that with metallic antimony as with different insoluble organic and inorganic compounds of antimony, so long as these contain antimony in the trivalent form, they can with certainty

cure mice, infected with nagana, dourine or *T. gambiense*, by a single injection. The mice, however, died of chronic poisoning after the absorption of metallic antimony and many other preparations of antimony. Various trivalent preparations of antimony are, however, relatively but slightly poisonous. Trixidin is of all known antimony preparations the most active on intramuscular injection; this is manifested by its chemotherapeutic coefficient (1:100), the permanent sterilisation which it causes, and by the absence of acute or chronic toxic effects. Animals infected with trypanosomes can be permanently sterilised in 100 per cent. of cases by one or two intramuscular injections of absolutely non-toxic doses of Trixidin. The question of antimony intolerance thus loses its significance for the treatment of chronic trypanosomiasis in men and the larger animals.

A large number of preparations, the therapeutic action of which was investigated by the authors, are classified as 'inactive,' 'active but not of practical utility,' and 'active and of practical utility.'

The second portion of the work is concerned with finding some means by which the poisonous action of chemotherapeutically active insoluble preparations of antimony, which lead on intramuscular injection to chronic poisoning, may be averted. The authors made use of a principle which has proved to be of great value in the treatment of syphilis, namely, the inunction of compounds in ointment form. They succeeded in curing infected mice by systematic, frequently repeated inunctions of metallic antimony in the form of ointments. In a proportion of cases relapses occurred. Trixidin is not active when administered by this method. With organic, insoluble compounds (*e.g.*, SCHEITLIN'S *Antimon-Antipyrin-Chlorverbindungen*) they succeeded in curing 60-70 per cent. of infected animals by this method. Acute and chronic poisonous symptoms do not follow inunction of insoluble antimony compounds which when injected intramuscularly or subcutaneously are toxic.

As a result of the second portion of their work the authors conclude that it is possible to cure a great proportion (66 per cent.) of the smaller animals (mice, rats, guineapigs, rabbits and monkeys) infected with dourine, nagana or *T. gambiense*, by inunction of metallic antimony or certain insoluble combinations, without the slightest toxic effect. In contradistinction to 'Therapia magna sterilisans' the authors designate this form of antimony treatment as 'Therapia mite curans.' The principle of the employment of insoluble organic preparations of antimony either in ointment form or through the formation of intramuscular depots may be suitable for the treatment of chronic trypanosomiasis in man or the larger animals.

The paper concludes with the statement that in Trixidin is found a remedy, which, in respect of its chemotherapeutic index and its permanent effect, far surpasses all others hitherto employed in the therapy of trypanosomiasis. The effect of the drug is to be tested in sleeping sickness patients and large animals infected with trypanosomiasis.

## IMMUNIZATION.

DARLING (S. T.). **The Immunization of Large Animals to a Pathogenic Trypanosome (*Trypanosoma hippicum* (Darling)) by Means of an Avirulent Strain.**—*Jl. Exper. Med.* 1913. May 1. Vol. 17. No. 5. pp. 582-586.

The object of this paper is to show that immunization of large animals can be effected by means of an avirulent strain of a pathogenic trypanosome (*T. hippicum*), that immunized animals completely recover from the infection and that they cannot be reinfected by the same strain nor by a virulent strain of the same species.

In a previous paper (*Sleeping Sickness Bulletin*, Vol. 4, p. 193) the author drew attention to the possibility of reducing the virulence of a strain of *T. hippicum* by selection from a guineapig.

Two mules, a dog, and a guineapig were inoculated from an infected guineapig which survived for the exceptionally long period of 336 days. These animals all recovered. Details are given of the two experiments with mules. The first mule was inoculated from the guineapig on the 279th day of the disease, and at the same time two other mules were inoculated with a virulent strain to serve as controls. The incubation period in the controls was 8 days whilst in the other animals it was 11 days. All three animals were treated with arsenic according to HOLMES'S method (*Sleeping Sickness Bulletin*, Vol. 2, p. 359). The controls died on the 44th and 105th day respectively. In spite of the arsenic medication trypanosomes remained in the first mule's blood for 6 months after the arsenic had been discontinued. The animal was definitely infected and passed through a typical attack of the disease, becoming very emaciated. Ultimately, it recovered and its condition improved until, 13 months after inoculation, auto-agglutination had disappeared and its blood inoculated into a white rat and a guineapig did not give rise to infection. The second mule was infected by means of *Musca domestica* (*Sleeping Sickness Bulletin*, Vol. 4, p. 182) with the same avirulent strain. The incubation period was prolonged to 10 days, showing that the strain was avirulent. [As the incubation period of the control animals was 8 days the increase in the mules infected with the avirulent strain was only 3 and 2 days respectively. Such a slight increase in the incubation period hardly appears sufficient to warrant this statement.] The disease ran much the same course as in the previous mule and the animal ultimately recovered. This animal was not treated medicinally.

One animal, specially tested, was observed to be immune to the same strain and resisted infection to a virulent strain as well.

The author concludes by stating that he believes that the production of subliminal infections by means of avirulent strains will yield efficient immunity against subsequent infection from a virulent strain, and the danger from a recrudescence of virulence within the immunized animal and the infection of other animals would appear to be nil.

W. Y.

## T. RHODESIENSE &amp; T. BRUCEI.

BEVAN (Ll.). Report on *Trypanosoma rhodesiense*. MS.—Report to the British South Africa Company. Dated March 20, 1913.

The author gives an account of the morphology of the parasite found in rabbits infected with a Zululand strain of *T. brucei*. The infected animals were sent to him from Pretoria. The parasite was polymorphic; typical long forms with free flagellum, intermediate forms, and short stumpy forms were seen. Among the latter were a few having the nucleus in the posterior third.

As the disease progressed, the posterior-nuclear forms increased in number and several were encountered with the nucleus actually posterior to the blepharoplast.

After calling attention to the fact that it was on this posterior displacement of the nucleus that STEPHENS and FANTHAM based their diagnosis of a new species of human trypanosome (*T. rhodesiense*), the author refers to the fact that YORKE and BLACKLOCK have observed similar forms in a strain of *T. equiperdum*, BLACKLOCK in a strain of *T. brucei* [Uganda], and WENYON in a strain of *T. pecaudi*. This feature then, in a strain of trypanosomes from the Luangwa Valley or Sebungwe District, can no longer be regarded as evidence of a new species.

[The Royal Society Commission has already described posterior nuclear forms in a strain of nagana obtained from Zululand (this *Bulletin*, Vol. I., p. 671). They consider *T. rhodesiense* (Stephens and Fantham) to be identical with *T. brucei* (Plimmer and Bradford). This, however, is doubtful as it has been shewn by STEPHENS and BLACKLOCK that *T. brucei* (Plimmer and Bradford) is a monomorphic parasite and easily distinguishable from either *T. rhodesiense* or *T. brucei* of the Uganda ox to which they have given the name *T. ugandae* (this *Bulletin*, Vol. I., p. 662). At the present time it would appear that if the name *T. rhodesiense* is to be dropped it must be in favour of *T. pecaudi*. As regards the presence of posterior nuclear forms in *T. rhodesiense*, Bevan seems to have revised his opinion, for in previous papers (*Sleeping Sickness Bulletin*, Vol. 3, p. 21 and Vol. 4, p. 214) he indicates that he had been unable to find these forms in this strain.]

W. Y.

LAVERAN (A.). Au sujet du *Trypanosoma rhodesiense* et du *Tr. brucei*.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 340-343.

The author quotes the statement of the Royal Society's Commission that "evidence is accumulating that *T. rhodesiense* and *T. brucei* (Plimmer and Bradford) are identical" (see this *Bulletin*, Vol. I., p. 661). These authors base this view chiefly on a study of the biometric curves of the two trypanosomes and on the fact that the nucleus is sometimes in the posterior position in *T. brucei* as in *T. rhodesiense*. LAVERAN holds that the conclusion of the Royal Society's Commission would be admissible only if they demonstrate (1) that the means of identification



employed by them are infallible to the extent that one can dispense with all others, (2) that the trypanosome named by them as *T. brucei* is in reality *T. brucei* (Plimmer and Bradford). Neither one nor the other of these postulates is above criticism. The author continues by discussing the value of biometric curves for the differentiation of trypanosomes. It is difficult to believe that the biometric method is infallible. DUKE has arrived at the conclusion that one can only accord to such curves a limited importance (see this *Bulletin*, Vol. 1, p. 137). The work of Muriel ROBERTSON on the cycle of *T. gambiense* in the vertebrate host (see this *Bulletin*, Vol. 1, p. 44) shows that curves of this trypanosome are likely to be dissimilar. Again it has been shown by WENYON and by LAVERAN and NATTAN-LARRIER that posterior nuclear forms occur in *T. pecaui* and by YORKE and BLACKLOCK that they are also encountered in a strain of *T. equiperdum*. The posterior position of the nucleus in certain individuals of *T. brucei* does not then warrant its identification with *T. rhodesiense*.

It is questionable if the trypanosome of Uganda which was used in the researches of BRUCE and his collaborators is identical with *T. brucei* (Plimmer and Bradford).

In his classification of the pathogenic trypanosomes (*Sleeping Sickness Bulletin*, Vol. 3, p. 356) the author has placed *T. brucei* among the monomorphic trypanosomes, whilst *T. rhodesiense* figures amongst the dimorphic.

STEPHENS and BLACKLOCK have recently shown that *T. brucei* of Zululand (Plimmer and Bradford) is monomorphic, whilst that from Uganda is dimorphic. They conclude that the two are not identical and propose for the Uganda parasite the name *T. ugandae* (see this *Bulletin*, Vol. 1, p. 662). If the Uganda trypanosome proves to be identical with *T. rhodesiense* as BRUCE and his colleagues consider, it is unnecessary to give it a new name, but LAVERAN believes that it will be necessary to prove the identity of the two and considers the method of cross immunization peculiarly applicable.

In conclusion reference is made to previous experiments of the author wherein he showed by cross immunization that *T. brucei* is different from *T. rhodesiense*. (*Sleeping Sickness Bulletin*, Vol. 4, p. 135.)

W. Y.

#### SOUTH AMERICAN TRYPANOSOMIASIS.

CHAGAS (Carlos). *Thyreoidite Parasitaria*. [Parasitic Thyroiditis.] —*Rev. Med. de S. Paulo*. 1912. Sept. 15. Vol. 15. No. 17. pp. 337-350. With 8 figs.

This paper was read before the Medical and Chirurgical Society of S. Paulo.

In his introductory remarks the author gives an account of the history of the disease, and of the experiments by which it was proved that *Schizotrypanum cruzi* is transmitted by the 'barbeiro,' as the insect is called in Minas Geraes. He points out that this bug has been wrongly included in the genus *Conorhinus*;

it really belongs to the genus *Triatoma*. The principal transmitter is *Triatoma megistus*; another is undoubtedly *Triatoma sordidus*. Recent investigations have shewn that *Triatoma geniculatum* transmits a parasite found in the armadillo which is supposed to be identical with *Schizotrypanum cruzi*.

The *Triatoma*, or 'barbeiro' as it is familiarly called, infests preferably those residences which are of primitive construction, and the walls of which have numerous chinks offering hiding places for the insect. Under favourable conditions they multiply prolifically, and are present in enormous numbers in houses which have been infested for some time. They transmit the disease during all three stages of their life cycle. The winged insect may live longer than a year. Its total life period including all stages is approximately two years. The insect lives in houses that are occupied. Empty houses are quickly abandoned by the *Triatoma* which migrate in search of food.

The parasite *Schizotrypanum cruzi* is found in the posterior intestine of the *Triatoma* either as Crithidial forms or as the adult trypanosomes; the presence of the latter is regarded as indicative of infectivity of the insect. There is some difficulty in demonstrating the presence of trypanosomes in the salivary glands, where they lie in the interstitial tissue between the glandular cells.

In the human organism *Schizotrypanum cruzi* is only found in the peripheral blood in any numbers during the acute stage of the disease. In chronic forms the parasite is found more abundantly in the tissues. Evolution of the parasite within the human body occurs in two ways, (1) the flagellate penetrates the tissues, especially the muscles, becomes rounded and undergoes successive binary division, (2) in the pulmonary tissue the parasite divides more regularly into eight equal units. The former of these is believed to be multiplication of the 'indifferent' forms, whilst the latter represents that of the 'sexual' forms. The young forms have at first only a nucleus and blepharoplast, later they become flagellated and get into the circulation. The forms from the lungs enter the erythrocytes and become intracellular parasites. The organs chiefly attacked by the parasites are the central nervous system, heart, suprarenals, testicles, uterus, ovaries, and striated muscle.

*Clinical and epidemiological observations.*—The disease is most frequent during the hot months (October to March), owing to the greater proliferation of the 'barbeiro' during this period. Only in children was the acute form observed: in adults the disease was chronic and was the result of infection acquired during childhood. The disease may be inherited—babies of from 15-20 days having been found to be infected. An autopsy performed on a foetus shewed macroscopic lesions typical of the infection.

The manifestations of the disease are multitudinous; clinically the cases can be divided into acute and chronic. The acute infection is characterised by high temperature and numerous trypanosomes in the peripheral blood, it is often complicated by intense meningo-encephalitis and in these cases death occurs in a few days. There is often a mucoid infiltration of the subcutaneous

tissue causing the facies typical of myxoedema. The thyroid gland is so constantly involved that the term parasitic thyroiditis has been used to describe the disease. The pulse rate is frequently diminished to 40 or in rare cases to 30 or even 20.

Parasitic foci and inflammatory processes of great intensity are seen in the central nervous system. Cerebral diplegia is common. As a rule, patients with diplegia are idiots. Infantilism is generally accompanied by complete idiocy, though cases were met with exhibiting only slight mental deficiency. Cases of infantilism are very common. [For further details CHAGAS's previous paper should be consulted (*Sleeping Sickness Bulletin*, Vol. 4, p. 341.)]

W. Y.

TRAMONTI (E.). *Alcune considerazioni sulla malattia di Carlo Chagas (Thyreoiditis parassitaria)*. [Some Observations on Chagas's Disease (Parasitic Thyroiditis).] — *Policlinico*. Sez. prat. 1913. May 18. Vol. 20. No. 20. pp. 697-701.

This paper is a review of the clinical features of schizotrypanosis. There is constant evidence in this disease of glandular insufficiency, on the part of the thyroid gland and suprarenals.

The clinical classification must depend on the predominant symptom, as the forms which the disease assumes are very various and not always sharply defined. The author divides the cases into two clinical categories, acute and chronic. Cases in which there are no acute morbid phenomena clinically, but in which the parasite is found in the peripheral blood by simple examination of fresh films, are grouped under acute. The acute form is subdivided thus:—(1) Those cases in which the nervous system is involved, *meningo-encephalitic* form. In this form the death rate is high and in any case the prognosis is unfavourable, paralysis, idiocy, and imbecility resulting. (2) Those in which the nervous system is not involved. Here the prognosis is better, the patient passing into a chronic state without serious impairment of function.

The chronic form is divided into five clinical groups—(a) *pseudomyxoedematous*, (b) *myxoedematous*, (c) *cardiac*, (d) *nervous*, (e) *chronic forms with acute crises*.

Mention is made of the group, *metaschizotrypanosis*, formed by CHAGAS, for those cases of infantilism, old goitre, and other morbid conditions consecutive to the disease.

Many cases of paralysis, aphaeresis and idiocy in children or infantilism in adults presented at the same time other clinical signs of Chagas' disease, while all endeavours to discover some other etiological factor such as syphilis or toxic, infective, or hereditary conditions, proved negative. The author finds from the evidence at autopsy that CHAGAS is justified in creating a 'nervous' form of schizotrypanosis. Multiple foci scattered in different parts of the brain reveal their presence clearly by clinical signs, so much so that one might say that Chagas's disease provides an excellent means of cerebral localization.

The most common motor disturbance is diplegia of cerebral origin, in which spastic symptoms predominate over paralytic.

The fact that parasitic foci are present on both sides of the brain or spinal cord explains the bilateral character of the paralysis. Athetotic and choreiform movements are frequently marked, the upper limbs being most affected and chiefly the fingers. The degree of loss of intelligence does not correspond to the amount of motor disturbance. Cases of complete idiocy shew sometimes very slight motor affection.

Speech is profoundly altered, total aphasia being common. Many cases present the clinical feature of pseudobulbar paralysis. Oculomotor paralyses occur, single muscles or several being affected. In cases of severe lesion of the nervous system general convulsions occur.

The author refers in some detail to two nervous cases in which post mortem examinations were made, one a child of three years, the other an adult of twenty-four, a complete idiot. In each case numerous parasites were found in the striped muscles. The author asks if it may not be possible that the endemic cretinism of the Alpine Valleys presents analogies in its aetiological mechanism to parasitic thyroiditis.

W. Y.

UNCLASSED.

LAVERAN (A.) & MARULLAZ (M.). *Au sujet du Trypanosoma talpae*.—*Compt. Rend. Soc. Biol.* 1913. May 16. Vol. 74. No. 17. pp. 1007-1008. With 1 text-figure.

The authors briefly review the previous literature on *T. talpae*. They examined 12 moles captured around Tournan (Seine-et-Marne), of which eight showed a very few trypanosomes while in one the parasites were not so rare.

The trypanosomes move too quickly to examine in detail in fresh preparations. In stained preparations only adult flagellates were found, which were  $25\mu$  to  $27\mu$  in total length and  $3.5\mu$  to  $4\mu$  in total breadth. The free portion of the flagellum is  $3.5\mu$  to  $4.5\mu$ . The undulating membrane is relatively straight. The posterior part of the body,  $6\mu$  to  $7\mu$  long, is drawn out in a spur-like manner. The blepharoplast is on one edge of the body, while the oval nucleus is on the opposite border. Chromophile granules occur in the cytoplasm. A text-figure indicates these characteristics.

The authors were unable to cultivate the trypanosome on a simplified Novy medium, and their attempts at inoculating the organism into 2 mice, 1 guinea-pig and 1 white rat were negative.

H. B. Fantham.

OGAWA (M.). *Studien über die Trypanosomen des Frosches*. [Studies on the Trypanosomes of Frogs.].—*Arch. f. Protistenkunde*. 1913. Apr. 19. Vol. 29. No. 2. pp. 248-258. With 1 plate and 3 text-figures.

The paper contains the results of Ogawa's work on *Trypanosoma rotatorium* of frogs. The main results of this, together with those of Frl. MENDELEFF-GOLDBERG and DOFLEIN, were set forth recently by the latter author (see this *Bulletin*, Vol. 2, p. 42).

Ogawa describes two main forms of the trypanosome: (1) "ribbed" (2) smooth, as observed both in the internal organs of the frog *Rana esculenta*, and in cultures. Full morphological details are given, cyclical variation in the condition of the nucleus being of the most interest. The formation of non-flagellate from flagellate forms by protoplasmic concentration, retraction of the undulating membrane and abandonment of the flagellum is described fully. Some stages of the parasite contain an enormous number of fat globules, their appearance suggesting that they are degeneration products. This is not the case as the fat is reserve food, and also these flagellates divide vigorously.

Large aggregation rosettes ( $100\mu$  diameter), the result of the intertwining of the flagella of many organisms, occur on both old and new cultures.

In conclusion, the author points out the possibility of demonstrating trypanosomes by cultural methods when they cannot be detected in the blood.

H. B. F.

PONSELLE (A.). *Technique pour la Coloration des Trypanosomes et Trypanoplasmes de Culture.*—*Compt. Rend. Soc. Biol.* 1913. May 23. Vol. 74. No. 18. pp. 1072-1073.

The technique, specially suitable for flagellates in culture, is as follows:

1. *Fixation.*—Pour on the dried smear a quantity of the following mixture, sufficient to cover it:—

Absolute alcohol, 50 cc.

Tincture of Iodine (of the French Pharmacopoeia), 10 drops.

Allow this to act for 5 minutes. Wash in absolute alcohol and leave to dry.

II. Then pour on the preparation some drops of serum, sufficient to cover it completely. Horse-serum, heated to  $56^{\circ}$ , serves very well. Leave for 5 minutes. Wash in distilled water.

III. Stain for 15 to 30 minutes with Giemsa solution diluted in the usual manner (1 drop to 1 cc. of neutral distilled water). Wash in distilled water. Dry.

H. B. F.

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## RELAPSING FEVER.

NICOLLE (Charles), BLAIZOT (L.), & CONSEIL (E.). *Etiologie de la Fièvre Récurrente. Son Mode de Transmission par les Poux.*—*Ann. Institut Pasteur.* 1913. Mar. 25. Vol. 27. No. 3. pp. 204-225.

During an epidemic of relapsing fever which raged in Tunis from February to August 1912, the authors were able to make a number of observations on this disease, which is usually very rare in Tunis. In this region relapsing fever has been known for a long time under the name of typhus, and both seem to be confounded in the native name "m'ard qmel," signifying louse disease.

Additional evidence in support of the view that relapsing fever is transmitted by the louse is afforded by the history of an outbreak during the last epidemic. A workman from Tripoli carried the infection to a "fondouk" in Tunis, of which two rooms were occupied by a colony of his compatriots. Shortly afterwards everyone of these persons became infected. As the result of a minute examination of the contents of the rooms, two fleas and one bug were found, and mosquitoes and Stomoxys were entirely absent. On the other hand, body and head-lice were present in enormous numbers.

The experiments recorded in the present article have been previously published elsewhere (see this *Bulletin*. Vol. 1, pp. 32-34).

E. Hindle.

KLEINE (F. K.) & ECKARD (B.). *Über die Lokalisation der Spirochäten in der Ruckfallfieberzecke (Ornithodoros moubata).* [On the Localisation of Spirochaetes in the Relapsing fever Tick.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. May 20. Vol. 74. No. 2. pp. 389-394.

The authors have examined the organs of forty-five females of *Ornithodoros moubata* obtained from a native hut on the shore of Lake Tanganyika. The ticks had not fed for two to three weeks. By microscopical examination of organ films twenty-three of these ticks were found to contain spirochaetes, the parasites occurring most frequently in the ovaries, and in descending order in the coxal glands, Malpighian tubules, cephalic glands, stomach, and salivary glands.

A number of ticks were fed singly on separate monkeys and afterwards examined for spirochaetes. Whenever a monkey became infected as a result of the bite, spirochaetes were found in the organs of the tick. The converse, however, was not true, for ticks were frequently found to contain spirochaetes without producing infection by their bite.

In another experiment monkeys were injected with the ovaries of single ticks, some of which had been used in the preceding series.

Combining the results of the two experiments it appears that out of eighteen ticks in which spirochaetes could be detected microscopically, thirteen were able to produce infection in monkeys, whilst out of nineteen ticks that were free from spirochaetes not one produced any infection. Therefore the presence of spirochaetes, as such, seems to be necessary in order to produce infection. The authors add that the parasites multiply by transverse division sometimes producing short forms. The presence of comma shaped bodies was frequently observed but in no case was any infection produced by injecting them into monkeys, and therefore they are not considered to represent any stage in the life-cycle of the spirochaete.

In addition the authors found spirochaetes present in a large proportion of the eggs laid by infected ticks.

E. H.

WITTRÖCK (O.). *Beitrag zur Biologie der Spirochaeta des Rückfallfiebers.* [The Biology of the Spirochaetes of Relapsing Fever.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. Apr. 25. Vol. 74. No. 1. pp. 55-60.

The author fed a number of "clean" first-stage nymphs of *Ornithodoros moubata* on monkeys infected with *Spirochaeta duttoni*. At intervals of one hour and 1, 2, 3, 4, 6, 10, 14, 18, 22, 24, 73, 75, and 96 days respectively, the contents of one or more of these ticks were inoculated into healthy monkeys which all became infected. The results show that there is no non-infective period of development corresponding with that of trypanosomes in the intermediate host, and the author considers that this furnishes additional support for the view that there is no relation between spirochaetes and trypanosomes.

In conclusion the author criticises the view that the "chromatin granules" in ticks represent any stage in the development of the spirochaete, for he has found these granules in the unfed offspring of uninfected *Ornithodoros*.

E. H.

**The Occurrence of Relapsing Fever in Somaliland.**—Received in Colonial Office May 12, 1913.

Dr. R. E. DRAKE-BROCKMAN, Medical Officer at Bulhar, records the outbreak of an epidemic of relapsing fever in Bulhar. In the "haffa" of Midzan, no less than fifty per cent. of the inhabitants were infected with the disease. Fortunately the mortality is said to be very low, but the incapacitating power of the disease is considerable owing to the number of relapses.

Large numbers of *Ornithodoros savignyi* were found in and around the infected "haffas," but no examples of *O. moubata*, and therefore the observer is of the opinion that the relapsing fever of this district is transmitted by the former species of tick.

[The occurrence of relapsing fever in Somaliland has been previously recorded by BRUMPT, who also showed that the infection could be transmitted by *Ornithodoros savignyi* as well as *O. moubata*.]

E. H.

LIAFFORGUE. *La Courbe Thermique dans la Fièvre Récurrente.*—*Bulls. et Mems. de la Soc. Med. des Hôpit. de Paris.* 1913. Mar. 13. 3 ser. Vol. 29. No. 9. pp. 579-581.

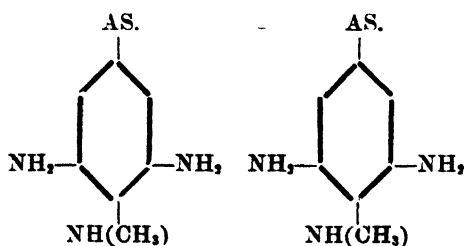
Amongst 22 cases of relapsing fever observed by the author in Tunis, eight showed very atypical temperature charts somewhat recalling "intermittent malaria." The patients presenting these atypical symptoms were all in a very bad state of health from other causes and it is suggested that this may have been the reason of the irregular character of the fever. Lafforgue points out that it is only possible to be certain of the diagnosis of relapsing fever by examination of the blood, for the temperature is liable to be uncertain.

In conclusion the author states that he and COMTE attempted to verify TIKTIN's suggestion that relapsing fever is transmitted by bugs, but their results have been constantly negative.

E. H.

GIEMSA (G.). *Beitrag zur Chemotherapie der Spirochätosen.* [Contribution to the Chemotherapy of Spirochaetoses.]—*Munchen. Med. Wochenschr.* 1913. May 27. Vol. 60. No. 21. pp. 1074-1078.

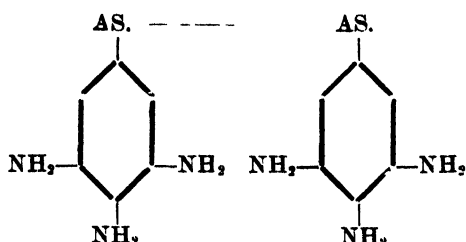
This article contains an account of the therapeutic action on certain spirochaetal infections of a new compound, *dimethyl-aminotetraminoarsenobenzol*, prepared by ACH and ROTHMANN in the laboratory of C. F. BÖHRINGER & SÖHNE and patented by this firm. This compound has the following constitution:—



and seems to combine a low toxicity with a very considerable therapeutic action. The author has tested its effect on infections of *S. gallinarum* in both fowls and canaries, *S. recurrentis* in mice, and syphilis in rabbits. The relation  $\frac{\text{dosis curativa}}{\text{dosis tolerata}}$  was found to be  $1/32$  for *S. gallinarum* in fowls,  $1/87.5$  for the same infection in canaries,  $1/1.56$  for *S. recurrentis* in mice, and  $1/12.7$  for syphilis in rabbits. These relations compare favourably with those of salvarsan (fowl spirochaetoses  $1/58$ , *recurrentis*  $1/2.7$ , rabbit syphilis,  $1/7$  to  $1/10$ ).



In conclusion the author discusses the relation between chemical constitution and therapeutic action. The introduction of methyl ( $\text{CH}_3$ ) groups into compounds has been found to lower the trypanocidal action, and therefore one would expect that hexaminoarsenobenzol—



—would be still more parasiticidal than the dimethyl derivative described above. Apparently this is the case, for the minimum effective dose of the latter compound is 0.011 gms. per kilo for rabbit syphilis, and 0.0014 gms. per 20 gm. mouse for *recurrentis*, whilst the corresponding effective doses of hexaminoarsenobenzolchlorhydrate are only 0.008 gm. and 0.00033 gm. respectively. No further information is given regarding this substance but the author states that he is continuing investigations on the subject. Other amino derivatives of arsenobenzol are in course of preparation and there seems every hope of still more effective compounds being obtained.

E. H.

BRONFENBRENNER (J.) & NOGUCHI (H.). On the Resistance of Various Spirochaetes in Cultures to the Action of Chemical and Physical Agents.—*Jl. of Pharmacology and Experimental Therapeutics*. 1913. Mar. Vol. 4. No. 4. pp. 333-339.

The authors have investigated the resistance of various spirochaetes and also *Bacillus coli* to the action of different chemical compounds and temperatures. The spirochaetes were all grown on solid media and before being exposed to the various substances were mixed with 0.9 per cent. saline. The following spirochaetes were employed:—*Treponema pallida* (heavy type), *T. pallida* (small type), *S. refringens*, *S. mucosum*, and *S. microdentium*; and the action of the following substances is recorded:  $\text{HgCl}_2$ ,  $\text{As}_2\text{O}_3$ , trikresol, phenol, saponin, sodium taurocholate,  $\text{NaOH}$ ,  $\text{HCl}$ , gentian violet, alcohol, salvarsan and neosalvarsan. Especial attention was paid to the effect of the last two substances because of their therapeutic importance.

The results are given in tabular form from which it appears that in these experiments the toxic effects exerted by the chemicals on spirochaetes are from twenty to one hundred times greater than on *B. coli*. The sterilizing property of salvarsan is practically the same as that of neosalvarsan; the toxic effects of the latter substance are increased from two to five times and possibly more in the presence of enzymes from the liver, and especially those from the blood.

Spirochaetes suspended in physiological salt solution are killed at a temperature of  $45^\circ \text{C}$ . in from seven to ten minutes.

E. H.

NICOLLE (Charles) & BLAIZOT (L.). **Deuxième Note sur la Courte Durée de l'Immunité dans le Fièvre Récurrente expérimentale.**—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 242-243.

The authors continue the history of a monkey that, after an interval of less than six months from its recovery from an attack of spirochaetosis, had been reinfected a second time by an inoculation of the same virus (see this *Bulletin*, Vol. 1, p. 621). Seventy-four days after recovery from this second attack, this monkey was again inoculated with the same virus and suffered from a third attack of spirochaetosis, showing that no marked immunity had been developed as a result of the two previous infections. This observation confirms the authors' view that the crossed immunity reaction is not a safe method of distinguishing the various races of the *recurrentis* group of spirochaetes.

E. H.

HIDAKA (S.). **Zur Frage der Beziehungen zwischen Syphilis und Recurrens-Immunität.** [The Question of the Relations between the Immunity against Syphilis and that against Relapsing Fever.]—*Zeitschr. f. Immunitätsforsch. u. experiment. Therapie.* 1913. Apr. 14. Vol. 17. No. 4. pp. 443-448.

The author has injected monkeys, infected with syphilis, with *Spirochaeta duttoni*, and also injected monkeys that had recovered from an attack of the latter infection, with syphilis, in order to determine whether the immunity was specific in each case. The results show that recovery from an attack of *S. duttoni* infection confers no immunity against syphilis, and conversely, animals infected with syphilis (in the so-called "Anergic" state of infection) are not immune against infection with *S. duttoni*.

E. H.

NAKANO (H.). **Über Immunisierungsversuche mit Spirochaeten-Reinkulturen.** [Studies on Immunity employing Cultures of Spirochaetes.]—*Arch. f. Dermatol u. Syphilis.* Orig. 1913. Mar. Vol. 116. No. 1. pp. 265-280.

Employing cultures of *Spirochaeta* (= *Treponema*) *pallida* the author has investigated certain immunity reactions of this organism, and his results are of interest for comparison with those obtained with the blood spirochaetes.

When spirochaetes from cultures, killed by heating to 60° C., were injected into rabbits, the serum of the latter was found to develop agglutinins and was active in dilutions of one in sixty to one in seventy, though not beyond. Precipitins could not be found.

With regard to the fixation of the complement, it was found very difficult with the various unspecific antigens that could be employed to decide whether the reaction was specific. Nevertheless there was a suggestion of the existence of such a reaction.

In the serum of the above mentioned rabbits, substances which dissolved spirochaetes were present in small quantity. In vitro, and in the sera of syphilitic human beings, such substances could not be detected.

An attempt to actively immunize rabbits by the injection of cultures of spirochaetes gave negative results and similar results were obtained on employing a vaccine prepared from human patients. The serum of these rabbits had a protective but not a curative action on rabbit syphilis.

Spirochaetes in the living body have a strong resistance against energetic cytolytic substances. Even after the injection of ten per cent. antiformin solution into a primary lesion in quantities sufficient to produce necrosis, living spirochaetes were still present.

E. H.

LAUNOY (L.) & LÉVY-BRUHL (M.). *Les Variations Numériques et Morphologiques des Globules Blancs chez les Poules infectées de Spirochaeta gallinarum.*—*Compt. Rend. Soc. Biol.* 1913. Apr. 18. Vol. 74. No. 13. pp. 754-756.

Fowls infected with *Spirochaeta gallinarum* were found to present fairly regularly two waves of hyperleucocytosis. The first is often very little marked and is coincident with the infection, whilst the second does not appear until the spirochaetes are disappearing from the blood. The two are separated by an interval of comparative leucopenia. The variations in the leucocytic formula are fairly constant; there being an excess of polynuclears during the infection, and an excess of mononuclears after the crisis. The polynuclears belong to two types: at first there is an excess of pseudo-eosinophiles "à bâtonnets," and later of neopolynuclears with spherical granulations.

Although these variations are present, they are not specific, since they may be found in fowls suffering from tuberculosis and also certain intoxications.

E. H.

GLEITSMANN. *Ueber die Beziehungen der Borreliën (Spirochäten) zu den Wirtszellen.* [On the Relation of Borreliæ (Spirochaetes) to the Cells of the Host.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Apr. 16. Vol. 68. No. 5-6. pp. 493-497. With 1 plate.

The first part of this paper is devoted to a criticism of Leonid FRÄNKEL's view (see this *Bulletin*, Vol. 1, p. 42) that spirochaetes are parasitic on the leucocytes. By means of the dark ground illumination the author has carefully examined the blood of fowls infected with *S. gallinarum* and mice infected with *S. recurrentis* and finds no evidence to support the view that the spirochaetes ever actively penetrate into the white cells. Occasionally they may be seen within the red corpuscles, but in every case the latter seem to be injured and Gleitsmann is of the opinion that the spirochaetes do not penetrate uninjured cells.

By means of BUCHANAN'S modification of the Levaditi-Yamamoto method, the author claims to have observed terminal flagella in *S. novyi*. The blood was not previously centrifuged, so therefore the appearances cannot be explained as the result of this process, as was the case in ZETTNOW'S preparations.

With regard to the nature of the swellings often seen along the length of the spirochaete and the mode of division the author does not come to any definite conclusions, but figures are given suggesting the occurrence of longitudinal division.

[The reviewer had previously expressed the opinion that Leonid FRÄNKEL'S theory was based on a study of artefacts (*loc. cit.*) and Gleitsmann's observations confirm this view. The so-called terminal flagella figured by the author seem to be merely the drawn out extremities of the pellicle of the spirochaete.]

E. H.

GROSS (J.). *Sporenbildung bei Cristispira*. — *Arch. f. Protistenkunde*. 1913. Apr. 19. Vol. 29. No. 2. pp. 277-292. With 1 plate.

The author describes the process of spore formation in *Cristispira tapetos*, a spirochaete which occurs in the crystalline style of *Tapes decussatus*.

The parasite merely breaks up into a number of short segments in a manner closely resembling the formation of coccoid bodies, or chromatic granules, in *Spirochaeta duttoni* and *S. gallinarum*. The latter part of the paper is devoted to a consideration of the nomenclature of the spirochaetes occurring in molluscs.

E. H.

## MALARIA.

BATES (John Pelham). **A Review of a Clinical Study of Malaria Fever in Panama.**—*Jl. Trop. Med. & Hyg.* 1913. May 15. Vol. 16. No. 10. pp. 145-153. With 3 charts.

The author points out that the teachings regarding the course and treatment of malaria by the best authors in text books as recent as 1905 are variable and somewhat confusing. During his stay in the Panama Canal Zone from 1904 until the present time over eight thousand cases of malaria (including all varieties) have passed under his notice. He found that under thorough quinine treatment the fever invariably subsided in one, two, or four days. Occasionally it did not subside till the fifth or very rarely till the sixth day. Consequently in 1907 he stated as an axiom that any fever that did not yield to quinine in five days was not malaria, or at any rate if a fever should continue for more than five days, with quinine properly administered, there was something other than malaria which was causing its continuance. ROGERS laid down a very similar axiom in 1908, and DEADERICK in 1909 also stated that four days was the maximum period of resistance to quinine. CRAIG in 1909 stated that the symptoms are easily controlled within a week although in rare instances the plasmodium may be very resistant to quinine and persist for eight to ten days. He advocates a dose of about two grammes (thirty grs.) daily. CASTELLANI and CHALMERS in 1910 however, while agreeing that four days of quinine treatment reduces the temperature and symptoms in cases of quartan and benign tertian, state that this is not always the case with regard to the subtertian (malignant tertian) fevers. They assert that they have met with cases in which the fever has remained unaffected while the parasites can be found in the peripheral blood, notwithstanding several weeks' quinine therapy by various methods. The author points out however that these authors are not quite definite as regards the amount of quinine which they administered in their resistant cases. In consequence he still holds to his axiom and thinks that there is insufficient evidence to support the belief that certain strains of malarial parasites are resistant to quinine. The continuance of fever in cases of malaria after four or five days of thorough quinine treatment (at least thirty grains daily) is due in his opinion to some underlying obscure disease such as kala azar, typhoid fever, or uncinarial anaemia. The latter is very liable to be confused with malaria on account of the irregular fever. HIRSCH pointed out that uncinarial anaemia was always looked upon as malarial cachexia until about 1885. Kala azar also was formerly considered as a kind of malarial cachexia. Three charts are given of cases of typhoid fever; in one of these malarial parasites were found. These disappeared under quinine treatment but the fever continued; a blood culture however gave a growth of *B. typhosus* and showed the true nature of the disease.

[This is a very excellent paper with a full list of references. Those interested should consult the original.]

D. Thomson

JAMES (W. M.). **The Canal Zone Treatment of Malaria.** [Correspondence.] — *Southern Med. J.* 1913. May. Vol. 6. No. 5. pp. 347-349.

The author states that in giving quinine in the treatment of malaria, there are two factors to be considered (1) The solubility and absorbability of the drug; (2) The amount of the drug that is taken up by the patient.

It is much better to give quinine in liquid form as it is then more readily absorbed than when in the form of pills and powders. This is important since the gastro-intestinal tract is often congested and irritated in acute malaria, so that its powers of absorption may be greatly diminished. Sometimes cases of malaria which are resistant to quinine given by the mouth may be rapidly cured by hypodermic administration of the same drug. He has found that malaria is often more difficult to cure by quinine treatment when it is complicated by certain conditions such as, anaemia, acute gastro-intestinal congestion, stasis, constipation, organic disease, tuberculosis, and especially syphilis. He states that the cause of relapses in malaria is not yet known with certainty, but the majority favour the view that they are due to the resistant forms of the asexual parasite. It is best therefore to treat a malarial attack vigorously with quinine, as small and insufficient doses do not completely kill all the parasites, but tend to favour the development of these resistant forms. This is why relapses so often occur after insufficient treatment with quinine in small or irregular doses. After eight years of experience in treating thousands of cases of malaria in Panama, the following is the routine method adopted:—

(1) Give a preliminary purge, three to five grains of calomel. This is followed in twelve hours by two ounces of a fifty per cent. solution of magnesium sulphate.

(2) An initial dose of twenty grains of a liquid preparation of quinine sulphate is given. (This preparation is made by dissolving five grains of quinine sulphate in one dram of distilled water with a drop of concentrated hydrochloric acid.)

(3) Fifteen grain doses of quinine (same preparation) three times daily are then given for a week, or for five or six days after the temperature is normal. The dose is then reduced to thirty grains daily and continued for ten more days. In Panama this treatment has proved much more satisfactory in preventing relapses than the former treatment of ten grains three times a day. If the infection is very severe the dose is increased to fifteen grains four times a day.

(4) When vomiting is present, quinine is given in doses of twenty-two and a half grains (1.5 grams) hypodermically. A French preparation of the bi-hydrochloride is used (obtained in ampoules). Each dose is given well diluted in twenty cc. of normal salt solution, and injected deep into the subcutaneous tissues with a large syringe. Abscesses are very rare after this treatment.

(5) The above treatment does not always prevent relapses, especially where there have been previous relapses due to insuffi-

cient treatment at the commencement. In such obstinate cases the author injects intravenously by means of a "606" apparatus, doses of one to two grams of quinine in 300-500 cc. of normal saline, on two succeeding days and then continues with forty-five grains a day by mouth as noted above.

(6) Tonics with arsenic, iron and strychnine are valuable adjuvants in treating the anaemia resulting from malaria. But small doses of quinine added to these have no curative value, but rather tend to increase the resistance of any remaining parasites,

D. T.

KIRK (William Redin). **The Early Recognition of Tuberculosis with Especial Reference to its Confusion with Malaria.**—*Southern Med. J.* 1913. May. Vol. 6. No. 5. pp. 300-303.

The author points out that early cases of tuberculosis which present few physical signs may be easily be mistaken for malaria in a malarious country, since in early cases of tuberculosis one finds occasional rises of temperature with sweating and anaemia. This mistake of course would not occur if the blood were examined carefully for the plasmodia, before quinine is administered. In order to avoid it he advises careful examinations of the blood at intervals. The temperature should be taken frequently. It is important to get a careful history of the case, and if possible one should employ the different tuberculin tests, examine the sputum, and make a very careful physical examination.

D. T.

VAN POOLE (G. M.). **A Case of Quinine Idiosyncrasy.**—*Military Surgeon.* 1913. Feb. Vol. 32. No. 2. p. 192.

The author describes a case of remarkable idiosyncrasy to quinine in a soldier. At 5.15 p.m. three-fifths of a gramme (rather less than ten grains) of quinine in tablet form was administered by mouth for prophylactic purposes. At 6.15 p.m. a telephone message was received stating that the patient was dying. He was hurriedly brought into hospital where it was found that he was in a comatose condition, from which he could not be aroused. His face was cyanosed. The radial pulse was very rapid and almost imperceptible. The pupils were widely dilated and acted very sluggishly. The breathing was shallow and stertorous, and the skin cold and clammy.

His stomach was washed out, heart stimulants were given hypodermically and heat was applied externally. At 7.15 p.m. he was still cyanosed but he could be roused by shaking. Pulse 118, pupils dilated but reacted readily, breathing slow and deep, temperature subnormal. He made an uneventful recovery.

Most authorities agree that in cases of pronounced cinchonism the heart's action becomes feeble and slow. In this case the pulse was rapid, but might have become slow later on, had he not been treated so promptly.

D. T

MÜHLENS. Bericht über eine Malariaexpedition nach Jerusalem. [Report of a Malaria Expedition to Jerusalem.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. May 3. Vol. 69. No. 1-2. pp. 41-85. With 6 plates and 5 text-figs.

Malarial investigation.—The total number of blood examinations was 7,921 from over 7,000 different people, *i.e.*, about one-tenth of the population of Jerusalem.

Of the 7,921 blood samples 2,071 contained malarial parasites, *i.e.*, 26.1 per cent. were infected.

The degree of infection varies greatly in the different districts of the town according to the class of population. In the inner Arab quarters, in the unhygienic districts, the infection amounts to 25-40 per cent. of all inhabitants; in the Jewish quarters within the city walls the infection is at least as high, if not higher. Similar percentages are obtained in the Jewish colonies outside the city walls. In the European settlements outside the city walls (not counting the native servants) 10-25 per cent. were infected; in Bethlehem 10-20 per cent. School children were found infected up to 37.4 per cent.

The species of parasite most frequently met with was the malignant tertian. Benign tertian was next in frequency, and quartan was less common. (In Bethlehem three quarters of all cases were tertian.)

Table I. illustrates the degree of infection among the different groups of the population.

Table II. and Fig. 3 illustrate the monthly distribution of the different species of parasites. The principal malarial season is from July to October.

During the five months of this expedition six cases of black-water fever occurred, four of which were seen by Mühlens himself.

Anopheles were found everywhere and in places were very numerous; they all belonged to the species *A. bifurcatus*.

Fig. 4 gives a record of the rainfall in Jerusalem. The propagation of the mosquitoes is in close relationship with the cistern method of water supply. The total number of cisterns in Jerusalem might be estimated at from 5,000 to 6,000 or even more.

*Preventive measures.*—No systematic anti-malarial measures had been taken previous to the author's arrival. General sanitary measures would in his opinion be also very good anti-malarial measures. The abolition of all cisterns, or even a general and efficient screening of the cisterns is regarded as impracticable on account of the enormous cost, not mentioning other great difficulties. Good results were partially obtained with quininization. Instruction in the schools, &c., is strongly advocated. The paper is well illustrated.



SEHRWALD (ERNST). **Zur Geschichte der Malariaübertragung.**  
 [Contribution to the history of the transmission of Malaria.]  
 —*München. Med. Wochenschr.* 1913. May 13. Vol. 60.  
 No. 19. p. 1040.

This is a brief account of STANLEY's efforts to prevent attacks of fever whilst in Africa. The author relates how STANLEY received the statement of EMIN Pasha that he had succeeded in preventing attacks of fever by the use of a mosquito net.

D. T.

#### PROPHYLAXIS.

HERMANT. **Fonctionnement du Service de Vente de la Quinine d'Etat dans la province de Nghê-An en 1912.**—*Bull. Soc. Med. Chir. de l'Indochine.* 1913. Apr. Vol. 4. No. 4. pp. 231-233.

In French Indo-China the state quinine is put up in tubes of ten tabloids, each containing 0.25 grammes of the sulphate. The price is 192 francs per 1,000 tubes. During 1912, 10,245 tubes, equal to 25 kilogrammes of quinine, were sold in the various depots. Beneficial results followed the distribution of the quinine, but the author gives no details or statistics.

D. T.

GIEMSA (G.). **Das Mückensprayverfahren im Dienste der Bekämpfung der Malaria und anderer durch Stechmücken übertragbarer Krankheiten.** [The Mosquito-spray Method in the Campaign against Malaria and other Mosquito-borne Diseases.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Mar. Vol. 17. No. 6. pp. 181-190.

The author describes certain improvements in the construction of the apparatus designed by him for the spraying of culicicide fluids. The composition of the fluid which he now advocates for use with his apparatus is as follows:—Pyrethrum tincture (20 parts of powdered pyrethrum blossom in 100 parts of 96 per cent. methylated spirit), 580 grams. Potash-soap (as free from odour as possible), 180 grams. Glycerin, 240 grams. For use dilute with twenty times its weight of water.

The fluid, which has been brought on the market in Germany as "Mückenfluid," causes hardly any damage to furniture or inconvenience to the operator. The cost of spraying a room of about 3,000 cubic feet is calculated to be approximately sixpence. Three figures illustrate different models of the apparatus.

D. T.

GRAHAM (J. D.). **Further Experiments in School Quininisation in the Districts of Meerut and Aligarh, 1911.**—pp. 22 + 7. With 2 maps and 30 tables. Report published by Local Government, United Provinces, India.

The author maintains that the quininisation of the school children in Meerut and Aligarh (India) during 1911 has produced results as successful as those in Muttra in 1909 and in

Budaun in 1910. He claims that this method of combating malaria is suitable for general adoption, but that the help of the district staff is required and also an attitude of sympathy on the part of the schoolmasters. The measure has been popular in all localities.

His statistics show that it causes a reduction of the splenic index as well as a reduction in the malaria rate. Large doses of quinine apparently produce a more rapid diminution in size of the spleen than smaller doses. He concludes that eighteen grains of quinine (sulphate tabloids) should be the minimum weekly dosage for the adult male community. Children are given doses in proportion to their age. The method of giving quinine twice weekly on 'alternate' days is apparently the most efficacious. A careful register should be kept wherever prophylaxis is being attempted.

D. T.

JACKSON (Thomas W.). **The Malarial Problem with Special Reference to Education and Diagnosis.**—*Interstate Med. Jl.* 1912. Nov. Vol. 19. No. 11. pp. 929-933.

The author points out that the statistical information regarding the true prevalence of malaria in the United States is not very reliable or accurate. The first reason of this is that in the average community deaths from malaria are not proved by microscopic blood examination, and again many latent cases which are perfectly capable of propagating the infection are missed. The only way to rectify this state of affairs is to educate further the physician as well as the laity, since the latter are still sceptical that malaria is propagated by the mosquito. He urges also the importance of making accurate sanitary surveys for malaria, as well as a blood census of the inhabitants and especially the children in malarious districts. Having obtained the desired data, efforts to cure the infected populace and to suppress the malaria-carrying mosquitoes should naturally follow. With regard to the infrequency of microscopic diagnosis of malaria in the United States, he makes the following statement:—

"During the year 1909, in a large laboratory connected with a metropolitan hospital, where more than twenty thousand new patients from all corners of the globe were treated and where a special feature is made of the teaching of malaria, only fifty eight of twelve thousand laboratory examinations made were blood examinations for malaria. This is something of an index of the failure of physicians generally to make use of the most direct and infallible diagnostic procedure in medicine."

D. T.

#### MONKEY MALARIA.

BLANCHARD (R.) & LANGERON (M.). **Le Paludisme des Macaques.** (*Plasmodium cynomolgi*, Mayer, 1907).—*Arch. de Parasit.* 1913. Mar. Vol. 15. No. 4. pp. 529-542. With 2 plates.

The authors state that it is very rare to find plasmodial parasites in monkeys bought in Europe. In 1911 however they bought a monkey (*Macacus cynomolgus*) which happened to harbour plasmodia in its blood. The infection in this monkey was mild.

Two other monkeys were inoculated subcutaneously with its blood, with the result that one of them died in twelve days from a very acute infection. The other showed only a mild and somewhat latent infection and lived a long time. In the first inoculated monkey the plasmodium appeared on the seventh day after inoculation, on the eighth day there were chiefly ring forms, on the ninth day they had become rosette bodies. On the tenth day the monkey was very ill and it was observed that the rosettes had broken up again into young ring parasites, very similar to the young rings of *Plasmodium falciparum* in man. They measured  $3\mu$  to  $5\mu$  in diameter. The containing red corpuscles develop granules resembling those of Schüffner in the case of benign tertian malaria. On the eleventh day the blood of the monkey showed marked anaemia and the parasites were found to have grown so large as almost completely to fill the containing red corpuscle. On the twelfth day the animal was dying, so it was killed in order to get the organs and blood in a fresh condition.

No young rings were found in the blood but various other stages of the parasites, from young compact globules to rosette forms. Some showed commencing segmentation with two, four and six chromatin masses while the rosettes showed eight, ten or thirteen merozoites. Pigment appeared in the small globular forms as a single grain, and became more plentiful as the parasites increased in size. Numerous pigmented leucocytes were found. The organs showed a much greater abundance of parasites than the blood, and it appeared that the capillaries were obstructed by considerable numbers of rosette forms. The spleen contained enormous numbers of parasites and great abundance of pigment, contained especially within the mononuclear cells. The endothelial cells of the liver also were loaded with pigment. The appearance corresponded exactly to that found in human malaria. This disease resembles therefore in almost all its aspects an attack of pernicious malaria in man. The authors consider that the parasite is identical with those which have been described by MAYER, FLU, MATHIS and LÉGER.

The corpuscles which contain the parasites remain normal in size, but invariably develop Schüffner's granules. The number of merozoites in the rosettes varies from eight to thirteen. This agrees with the finding in MAYER's parasite, but the parasite of MATHIS and LÉGER appeared to form, almost always, sixteen merozoites.

The authors also observed the presence of gametes. The macrogametes were large globular parasites, filling the corpuscle almost completely. The protoplasm had an intense blue colour, the nucleus was generally excentric and stained a deep red colour. They contained abundant pigment, very black and in large grains. The microgametes were also large and globular, the cytoplasm stained a feeble rose or violet colour, while the nucleus which is large stains a pale rose colour. MAYER has observed that the gametes appear in the blood two days after the verification of the presence of parasites from an inoculation. The duration of the cycle of schizogony in this parasite is forty-eight hours.

In their conclusions, the authors state that this parasite observed by them in the *Macacus cynomolgus* is identical with the *Plasmodium cynomolgi*, MAYER, 1907. It is a distinct species, differing from the *Plasmodium inui* of HALBERSTÄDTER and PROWAZEK, 1907, in the presence of Schüffner's granules and the form of the pigment. *Plasmodium cynomolgi* may be very pathogenic to the *Macacus cynomolgus* when it is inoculated into an animal which has not been immunised by a former attack. This pathogenic action takes the form of pernicious attacks of fever, accompanied by a heavy parasitic infection of the whole circulation. The pathological lesions produced are similar to those observed in man in pernicious malaria.

The article is illustrated by two coloured plates.

D. T.

BLANCHARD (R.) & LANGERON (M.). **Nouvelles Recherches sur le Paludisme des Macaques d'après les Notes Posthumes de Xavier Bouniol.**—*Arch. de Parasit.* 1913. Mar. 12. Vol. 15. No. 4. pp. 599-607. With 1 coloured plate.

The authors state that BOUNIOL conducted a research on eight monkeys (*Macacus cynomolgus*) which were infected with *Plasmodium cynomolgi*. He unfortunately died at the end of a year's work before he had published his results.

The authors were, however, able to obtain most of his notes and give an account of his work, as gleaned from these. Of the eight monkeys employed by BOUNIOL four had their spleen removed. In two of these the splenectomy resulted in an acute exacerbation of the disease with increase of the plasmodia and death. In the other two the disease remained chronic, death taking place from an intercurrent disease after some months. The most interesting fact brought out in Bouniol's research is the effect of the splenectomy on the morphology of the parasite. Splenectomy resulted in the appearance of very amoeboid forms of the plasmodia, in which the protoplasm and chromatin were drawn out into long filaments. It also resulted in the production of an increased number of merozoites; as many as sixteen were produced instead of the usual eight. The other notable facts brought out by his research were, the constancy of the forty-eight hours' duration of the asexual cycle of the parasite, the constant appearance of Schüffner's granules in the infected red corpuscles, the variable pathogenic effect of this parasite on the infected monkeys, the rise of the temperature at the end of the infection, and the sub-normal temperature shortly before death.

D. T.

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## BLACKWATER FEVER.

**AFRICA. Reports on Blackwater Fever in the Tropical African Dependencies.**—45 pp. With 2 maps. F'cap. 1912.  
London: Printed by H.M. Stationery Office.

The Secretary of State, on the suggestion of the Advisory Medical and Sanitary Committee for Tropical Africa, has circularised the Governors of the various East and West African colonies as regards the prevalence of blackwater fever in their respective territories. At present the conditions favouring the incidence of blackwater fever are not thoroughly understood and it has been considered that the collation and careful study of all cases which occur might throw some light upon the etiology of the disease and might suggest means for preventing or diminishing its occurrence. With this object the Secretary of State has requested that a special report may be forwarded annually by the Principal Medical Officer on all cases of blackwater fever occurring within the year in the colony, attention being particularly paid to the following points:—

**I. *Locality*:**

“(a) Physical features (*e.g.*, swamps, bush, forest, &c.).

“(b) Occurrence of a series of cases in any one place, particularly in any one building, specifying dates and relation to native dwellings and intercourse.

“(c) Insect fauna; particularly biting or sucking insects such as mosquitoes and biting flies, ticks, bugs, lice, fleas, &c. Specimens should be obtained and identified where possible.

**II. *Seasonal Variation*:**

(a) Marked or unusual climatic conditions.

**III. *Personal History*:**

“(a) Medical history of patient (*e.g.*, previous diseases, attacks of malaria, habits regarding quinine-taking, &c.).

“(b) Previous movements of patient and personal conditions to which he has been subject.

“(c) Microscopic examination of the blood (noting relation of examination to stage of illness).”

Replies have been received from the Gambia, Sierra Leone, East Africa, Nyasaland, Uganda, the Gold Coast and Northern Nigeria. In addition to giving answers to the questions, some of the medical officers have written short accounts of their own personal views on the etiology of the condition and some have given clinical details of certain cases. Many of these are interesting and instructive.

An analysis of the answers given to the questions asked brings out very strongly the fact of previous attacks of malaria preceding the onset of blackwater fever. People who have suffered from many attacks of malarial fever and are also irregular in the taking of quinine are, without doubt, those who are attacked, but even regular quinine takers, if they have suffered much from malaria, may suffer, as the following cases show:—

An Asiatic suffered occasional attacks of malaria which, however were very mild. He took quinine very regularly. Blood examination in the attack showed a few malarial parasites of the tertian variety. These had disappeared from the blood by the

third day of the illness. His attack of blackwater fever was a mild one, recovery taking place about the tenth day.

In another case of blackwater fever, treated in hospital at Bathurst, the patient is said to have taken a regular dose of five grains of quinine per diem. He had been for three previous tours on the West Coast. In his first tour he contracted malaria and since that date suffered from slight attacks, those generally not lasting more than 22 hours. The attack of blackwater fever came on suddenly with violent vomiting and diarrhoea. The exciting cause seems to have been a dose of five grains of calomel taken that evening.

All FORDE's blackwater fever cases, without exception, occurred in persons of some length of service in West Africa who had never taken quinine regularly as a prophylactic, but who, when they found malarial fever coming on, generally took two or three large doses of the drug and as soon as the fever had left them took no more until their next attack.

MANNING says that there appears to be little doubt that those not using the daily ration of quinine are far more liable to the disease; in other words, haemoglobinuric fever is a manifestation of malarial toxicity.

Again, HODGES, speaking of Uganda, says that his reports appear to show a definite relation between blackwater fever and malarial infection, while in the medical case sheets of the other cases reported, the same thing is seen, viz.:—many attacks of malarial fever.

As regards the other points of information asked for in the circular, little can be deduced from the answers so far given, but the cases reported upon are comparatively few up to the present date. The insect fauna seems to be much the same wherever the cases occur and the same may be said of the physical features of the country in respect to swamps, bush, &c. The typical type of country for blackwater fever is apparently similar to that found where malaria prevails. The increasing frequency of the disease is due no doubt to the increase of susceptible individuals, *i.e.*, the increased white population which yearly goes to the endemic areas.

G. C. Low.

STANNUS (H. Stannus). **The Etiology of Blackwater Fever.**—*Trans. Soc. Trop. Med. & Hyg.* 1913. Apr. Vol. 6. No. 5. p. 181.

The author states that during eight years in Nyasaland he has never seen blackwater fever in a native. His experience leads him to believe that in the absence of malaria there is no blackwater fever. He has seen no case of haemoglobinuric fever in which there had not been a previous attack of malaria treated by quinine. Several patients seen by the author have had three and four attacks of this disease.

D. Thomson.

NEWELL (A. G.). **The Etiology of Blackwater Fever.**—*Trans. Soc. Trop. Med. & Hyg.* 1913. Apr. Vol. 6. No. 5. pp. 177-181.

The author believes firmly in the relationship between blackwater fever and malignant tertian malaria. Quinine is not a cause of blackwater fever but may help to induce it at a time when the liver is extremely congested. In his opinion the haemoglobinuria is due to the intense congestion of the liver, caused by the malignant tertian parasites. If the condition of the liver is observed before giving quinine and if sufficient quinine is given regularly in intensely malarious districts there should be no blackwater fever. He agrees with CHALMERS that there is a quinine haemoglobinuria and a malarial haemoglobinuria.

D. T.

COLES (Alfred C.). **Protozoal-like Structures in the Blood in a Case of Blackwater Fever.**—*Lancet.* 1913. May 3. pp. 1230-1232. With 1 text-figure.

The author made prolonged and repeated examinations of numerous films of blood, taken at all stages during a fatal attack of blackwater fever in a man age 28 who had just returned from Rhodesia. He was unable to find any malarial parasites or pigmented leucocytes, and no spirochaetes could be detected in fresh specimens under the dark ground illumination apparatus. In some of the blood films taken towards the latter stages of the disease a few peculiar granular bodies were found. The earliest period at which one of these bodies was seen was on the fourth day of fever, and then only a single example was found, but they were more frequent on the last day. These bodies were always exceedingly rare, about 4 were found in each film on the average; they were nearly always spherical, with a very definite outline. The protoplasm varied in tint from pale blue to bluish red. The diameter varied from  $5\mu$  to  $17\mu$ . They did not possess a nucleus, but contained within the protoplasm a number of small round or oval dots which stained a deep chromatin red colour. The number of dots found in each cell varied from 40 to 200. They measured  $0.3\mu$  to  $0.75\mu$  in diameter. Similar bodies were met with in the protoplasm of a few very large mononuclear leucocytes. The blood of the patient showed a large number of nucleated red cells and the haemoglobin amounted only to 14 per cent.

D. T.

HAND (Albert). **Malarial Hemoglobinuria—Report of Case.**—*Southern Med. J.* 1913. Mar. Vol. 6. No. 3. p. 171.

The author describes a case of malarial haemoglobinuria in a white girl aged 9 years. She had suffered from chronic malaria with anaemia and cachexia for two years and had had a previous haemoglobinuric attack five months before. After a malarial paroxysm two days previously the patient had a severe chill with immediate haemoglobinuria. This attack was severe and typical

with marked anaemia and severe vomiting. No parasites could be detected in the blood. The haemoglobin fell to 10 per cent., she became semi comatose, and developed a general oedema and paralysis of the right arm. Quinine was given in moderate doses, about six grains every twelve hours and under this treatment the urine cleared up. During the attack the blood showed a marked leucocytosis amounting to 80,000 white cells per cmm. The anaemia was very severe with 10 per cent. haemoglobin and 500,000 red cells per cmm. The transient paralysis was probably due to a localised oedema of the brain, a part of the general oedema. The patient appears to have recovered.

In the former haemoglobinuric attack quinine had been given six hours previously, but none had been given for fourteen days before the onset of the attack described.

D. T.

**STANNUS** (Hugh Stannus). **The Treatment of Suppression in Blackwater Fever.**—*Jl. Trop. Med. & Hyg.* 1913. May 1. Vol. 16. No. 9. pp. 131-133. With 1 chart.

The author describes a case of blackwater fever in a European aged twenty-five in Nyasaland, in which there was complete suppression of urine.

He tried every measure possible to relieve this suppression without success and, as a last resort, operated on the left kidney, incising the capsule from pole to pole and the middle two-thirds of the organ itself. The result was that small amounts of urine (two and a half to four ozs.) were passed daily during the next four days. The patient however died. The author thinks it is probable that the patient might have recovered had the operation been performed earlier. All the cases of blackwater with complete suppression which he has known in Nyasaland have died without any sign of the suppression being relieved in the least degree. In such cases he wonders if it would be justifiable to operate early, since good results have been obtained by this procedure in cases of suppression in acute nephritis.

D. T.

**DA MATTA** (Alfredo Augusto). **A Febre Biliosa Hemoglobinurica no Amazonas e o seu Tratamento pela Cecropia.** [Bilious Haemoglobinuric fever in Amazonas, Brazil, and its Treatment with 'Cecropia.'].—*Rev. Med. de S. Paulo.* 1912. Sept. 30. Vol. 15. No. 18. pp. 357-364. With 4 charts.

Blackwater fever is frequent in the Province of Amazonas. In all the cases haemoglobinuria was in evidence, though in varying degrees of intensity, the colour of the urine ranging from port wine colour to black. The author, whose cases numbered altogether 35, made the following observations:-

(1) Cases occurred after only a few months' residence in Amazonas. (2) There was a history of previous malaria in some form or other. (3) The absence of malarial parasites in certain phases of the disease was noted. (4) Pronounced anaemia leading to a leucopenia in the final stages of typical cases was evident.



The author explains the presence of haemoglobin in the urine, quoting SIMPSON and ROSS. His observations are in accordance with those of DEADERICK, who distinguished three phases, viz., (1) The attack is like one of malarial fever with parasites present in the blood and no haemoglobinuria. (2) Fever may be present, the parasites being generally absent, and haemoglobinuria appears. (During this phase the author made 24 blood examinations—all negative). (3) The post-haemoglobinuric stage with clear urine and fever, which if present is similar to that of a secondary attack of malaria, and generally no parasites are found in the peripheral blood. (The febrile attack during this phase was observed by the author in only three out of 35 cases.)

Quinine may be the cause of an attack of haemoglobinuria. The author found that in such cases haemoglobinuria sets in within three to six hours after its administration; beyond this time quinine is not responsible. The diazo-reaction and reactions of GMELIN and GRIMBERT are positive during the third stage.

The author found the best treatment to be the serum treatment of GOUZIEN. He also gives calomel, Vichy water, and lactate or chloride of calcium. The calcium salts, however, must not be continued for any length of time, as they will then give results contrary to those desired. 'Cecropia'\* is considered invaluable. It eliminates bile and is a powerful diuretic. No bad effects were experienced even after a dose of 25 grams of the liquid extract.

Four charts are given.—Chart I is of a fatal case with *Plasmodium praecox* in the blood and intense albuminuria. It is not stated at which stage of the disease the parasites were present in this case. Charts II. and III. showed negative blood findings on the day before the onset of blackwater. Chart IV. is that of a case with *Pl. praecox* in the blood on the day before onset.

D. T.

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\* *Cecropia palmata*, *C. peltata*, *C. baraensis* and other species are mentioned as occurring abundantly in Amazonas. It is not stated from which species the alkaloid (in the author's opinion a glucoside) is obtained.

## TROPICAL DISEASES BUREAU.

TROPICAL DISEASES  
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## DYSENTERY.

## AMOEBIC DYSENTERY.

CONOR (A.). *La Dysenterie Amibienne en Tunisie.*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 316-317.

The author after acknowledging that NICOLLE and CATHOIRE showed that the bacillary dysentery in Tunis is due to *B. dysenteriae* Shiga points out that there is also present in that country a dysentery due to entamoebae, which in the author's opinion are more often *E. histolytica* than *E. tetragena*.

S. R. Douglas.

CHAUFFARD. *La Dysenterie Amibienne chronique.*—*Presse Méd.* 1913. May 14. Vol. 21. No. 39. pp. 389-391.

The report of a clinical lecture on chronic amoebic dysentery given at the medical clinic of Saint Antoine.

The lecturer in describing the life history of the *Entamoeba* causing dysentery follows the description given by SCHAUDINN of *E. histolytica*; he still believes that the pathogenic amoebae can be cultivated on artificial media.

A long account is given of the morbid anatomy found at post-mortem examinations. The lecture concludes with the detailed account of a case of chronic dysentery treated by subcutaneous injections of emetine; this case has already been referred to in this *Bulletin*, Vol. I., p. 714.

S. R. D.

DESSY (S.) & MAROTTA (R. A.). *Contribución al Tratamiento de la Enteritis Disentérica y del Absceso del Hígado (amibiano), con el Método de Rogers.* [Contribution to the Treatment of Amoebic Dysentery and Liver Abscess by Rogers's Method.] —*Semana Médica.* 1913. Apr. 3. Vol. 20. No. 14. pp. 797-799.

An account of the successful treatment of a case of amoebic dysentery by injections of emetine.

The patient, a male aged twenty-five years, had suffered for a year with symptoms of colitis of a dysenteric type, and had tried all kinds of treatment without relief. Tenderness and burning sensations were present over the whole course of the large intestine, and the motions were numerous up to twenty-three a day, liquid and containing large quantities of pink mucus. Microscopic examination of the stools showed numerous amoebae.

A first injection of two centigrammes of hydrochloride of emetine was given subcutaneously, and on the following day the patient declared that he had the first good night since the commencement of his illness. The local symptoms were less and the motions fewer. A second injection of the same amount was then given, and on the third day he declared that he felt quite well. There had been no motion since the previous day, and palpation over the colon caused no pain. The appetite began to return. A third injection of two centigrammes was given, and at six p.m. of the same day the first solid motion was passed. No amoebae could be detected in it, either alive or dead. A fourth injection of the same amount was given on the following day, by way of precaution, but the patient seemed to be definitely cured, the bowels thenceforth acting normally.

S. R. D.

MARCHOUX (E.). *Le Chlorhydrate d'Éméline dans la Dysenterie Amibienne.*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 313-316.

The author reports two cases treated with emetine hydrochloride given by subcutaneous injection in whom, although all symptoms of dysentery disappeared, amoebae or cysts could be demonstrated in the stools. From observations made in these cases the author concludes that the amoebae present in the intestine are not invariably exterminated and that in some of the cases treated with subcutaneous injections of emetine salts we may expect relapses.

S. R. D.

DUFOUR (Henri) & THIERS (J.). *Dysenterie Chronique Amibienne traitée par le Chlorhydrate d'Éméline. Présentation de Malade.* —*Bull. et Mém. Soc. Méd. des Hôpitaux de Paris.* 1913 Apr. 24. 3 ser. Vol. 29. No. 13. pp. 827-829.

The report of a case of chronic dysentery contracted in Portuguese India treated by the subcutaneous injection of hydrochloride of emetine. Four to six centigrams were administered per diem. Recovery was rapid.

S. R. D.

LYONS (Randolph). **The Treatment of Amebic Dysentery with Subcutaneous Injections of Emetine Hydrochloride. Report of Six Cases.**—*Jl. Amer. Med. Assoc.* 1913. Apr. 19. Vol. 60. No. 16. pp. 1216-1220.

An account of six cases of dysentery treated with subcutaneous injections of emetine hydrochloride. There was one fatal case. The doses of emetine given were from one half to three quarters of a grain per diem. The average time before the stools became normal was nine days. The author concludes by saying that up to the present none of the cases has shown signs of relapse.

S. R. D.

i. DARLING (Samuel T.). **Budding and other Forms in Trophozoites of *Entamoeba tetragena* simulating the "Spore Cyst" Forms attributed to "*Entamoeba histolytica*."**—*Arch. Internal Med.* 1913. May 15. Vol. 11. No. 5. pp. 495-506. With 3 plates.

ii. **The Identification of the Pathogenic *Entamoeba* of Panama.**—*Ann. Trop. Med. & Parasit.* 1913. June 10. Vol. 7. No. 2. pp. 321-329.

i. Several papers by Darling on the identity and life-cycle of the amoeba causing amoebic dysentery in Panama have been reviewed recently (see this *Bulletin*, Vol. 1., pp. 180, 462, 720). In this paper (i) the results of further studies are set forth.

The author's conclusions are as follows:—

"I have seen in fresh preparations of *E. tetragena* from a fatal case of dysentery bizarre pseudopodia and buds with refractile bodies in the extremities, though I did not observe extrusion of buds or nuclei. If one had merely observed in fresh preparations alone from a case of dysentery in man, the changes corresponding to those seen in stained preparations, the impression received would no doubt have been very much like that described by Schaudinn and Craig as the spore cyst formation of *E. histolytica*; but from an examination of the far more richly infected material from the kitten 'which occurs after a lengthy period of lively increase,' the true nature of the budding forms is understood and the opinion is formed that these changes are analogous to such essentially pathological manifestations of cellular degeneration as karyolysis, karyorrhexis, pyknosis and dislocation and extrusion of nuclei; moreover, the budding process is analogous to changes seen in mononuclear metazoal cells; for example, in defunct plasma cells or lymphocytes in the blood-stream, lymph-nodes and other locations.

"The descriptions of the life-cycle of *E. histolytica* by Schaudinn and Craig, therefore, are in all likelihood those of a spurious species, having resulted from observations of pathological changes in senile races of *E. tetragena*."

The figures illustrate clearly the phenomenon described.

ii. In the second paper the author describes (a) the methods of examination used by him (he recommends staining *intra vitam* with gentian violet), (b) the amoebae as they appear in fresh and in stained preparations and (c) feeding and inoculation experiments.

The occurrence of four-nucleated schizonts is definitely mentioned. The author remarks that "when tetragena cysts have been fed to half-grown cats there has resulted not the typical

entamoebic colitis, such as is usually described in the literature, but an enteritis, and in this lesion in the ileum trophozoites have been found which, though arising from *tetragena* cysts, had the morphology not only of *E. tetragena* but of *E. histolytica* and *E. nipponica*."

[The use by Darling of the remark that *E. histolytica* is a "spurious species" is unfortunate and very probably incorrect. Recent research strongly suggests that the life-cycle of *E. histolytica* described by SCHAUDINN is inaccurate as regards the peculiar exogenous cycle—probably degenerative and associated with minute spores—recorded by him. The correct reproductive cycle of *E. histolytica* seems rather to be the formation of four nucleate cysts, as discovered by VIERECK for *E. tetragena*. If *E. histolytica* and *E. tetragena* are identical, then the name *E. histolytica* must stand, but its life-cycle would be not that recorded by SCHAUDINN but that first described by VIERECK, and the species name *tetragena* must then drop. At present however, it would be presumptuous to dogmatise on such a difficult subject.]

H. B. Fantham.

CRAIG (Charles F.). *The Identity of Entamoeba histolytica and Entamoeba tetragena. A Preliminary Note.*—*Jl. Amer. Med. Assoc.* 1913. May 3. Vol. 60. No. 18. pp. 1353-1354.

Previously Craig considered *E. histolytica* and *E. tetragena* to be distinct, but now he holds that *E. tetragena* is identical with *E. histolytica*, and that the life-cycle of the latter, described by SCHAUDINN and confirmed by himself and others, is incorrect so far as reproduction by budding or spore-formation is concerned.

His present opinion is based on the study of preparations sent by Dr. W. M. JAMES from three cases of histolytica-infection occurring in Panama, and on the study of a case of histolytica-infection in an inmate of the Soldiers' Home at Washington who had contracted dysentery in the Philippine Islands. Regarding the examination of these preparations Craig writes: "In all these cases I have been able to observe every form of both *histolytica* and *tetragena*, so far as nuclear structure and reproduction goes, that has been described for either species, and as double infection in these cases can practically be ruled out, I am forced to conclude that there is only one species of entamoeba present and that all of the forms found belong to the life-cycle of *Entamoeba histolytica*." He now considers that the budding forms supposed to represent a stage in the reproduction of *E. histolytica* are degenerative in character, and that the normal method of reproduction of *E. histolytica* in the cystic stage is by the formation of cysts containing four daughter nuclei.

As *E. tetragena* is, so far as present evidence goes, identical with *E. histolytica*, the name "*tetragena*" as a specific name should be abandoned. Craig and JAMES have observed that the amoebae presenting the typical histolytica nucleus do not as a rule show cysts. Craig does not agree with DARLING that *E.*

*histolytica* is a "spurious" species, as "amending the description of a species does not warrant the use of a new specific name." [See remarks above.]

H. B. F.

ORNSTEIN (Otto). *Zur Ätiologie der Amöbenruhr*. [Etiology of Amoebic Dysentery.] — *Arch. f. Protistenkunde*. 1913. Mar. 22. Vol. 29. No. 1. pp. 78-83. With 10 text-figures.

The material used came from El Tor, in the Sinai Peninsula, which is sometimes a quarantine station for the pilgrims going to Mecca.

From examination of fresh material it was concluded that the causative agent of the amoebic dysentery at El Tor was *E. histolytica*. Small cyst forms, as described by SCHAUDINN, were seen. Few of the amoebae showed compact nuclei, but contained chromidia.

The author points out that amoebae which might be interpreted as division forms probably result from plasmogamy, that is, from the apposition of degenerating amoebae.

After examining stained preparations, the author concludes that budding is an expression of degeneration division, as observed at the beginning of cyst formation in *E. tetragena*. HARTMANN, describing degeneration forms of *E. tetragena* (such as occur at the beginning of cyst formation), pointed out that KOIDZUMI in describing *E. nipponica* was dealing with a degenerating *E. tetragena*.

Cysts were rarely observed.

The large number of degenerating amoebae seen is probably to be accounted for by the fact that the patients had been treated before the material for examination was collected, and possibly the treatment had affected the amoebae adversely.

H. B. F.

#### LIVER ABSCESS.

SAMBU' (Edouard). *Notes Cliniques sur les Abscès du Foie au Tonkin*. — *Ann. d'Hyg. et Méd. Coloniales*. 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 48-103.

The author in a long paper analyses with considerable skill his experience of this disease in Tonkin, where he had worked for some ten years and had seen over 100 cases.

His paper is divided into the following parts:—

#### Part I. Etiology.—

Under this heading the most noteworthy points are:—

- (a) The rarity of the disease amongst the Annamites; for instance, in the hospital at Haiphong 111 cases were treated in thirteen and a quarter years, of whom 103 were Europeans. Again in the town of Hanoi in the year 1910 out of a total of 75 deaths amongst 12,534 Europeans twelve were certified as due to liver abscess, while of the 1,776 deaths occurring in over 62,000 natives only three were from this disease.

- (b) Liver abscess was nearly always consecutive to dysentery and a definite history of this disease could be obtained in 64 per cent. of the cases; further, from the study of fatal cases the author concludes that the so-called chronic diarrhoea of Tonkin is in reality masked dysentery and, taking this into account, the percentage should be placed at a much higher figure.
- (c) Alcoholism and opium smoking the author believes to be important factors in diminishing the resistance of the organism and thus rendering the individual more liable to the disease.

## Part 2. Notes on the pathological anatomy.—

### Number of abscesses.

In 56 cases there was	...	...	1 abscess.
„ 11 „ „ „	...	...	2 abscesses.
„ 10 „ „ „	...	...	3 „
„ 2 „ „ „	...	...	4 „
„ 1 „ „ „	...	...	5 „
„ 1 „ „ „	...	...	8 „
„ 6 „ „ „	more	than	8 „
„ 2 „ „ „	at	least	3 „

or in 89 cases in 56 (=63 per cent.) there was a single abscess and in 33 (=37 per cent.) there was more than one.

### Situation of the abscess.—

	Per cent.
Right lobe	77·5
Left lobe	19·38
Spigel's lobe	2·32
Quadrant lobe	·80

### Volume of abscess.—

Several litres	7
About 1 litre	23
More than 500 grams	27
About the size of an orange	28
Miliary	6

## Part 3. Diagnosis of liver abscess.—

The author very strongly argues in favour of exploratory puncture, in which as he states he is supported by all practitioners of wide experience.

## Part 4. Treatment.—

Early opening by means of an extensive resection of a rib if situated as is usual under the cover of the ribs.

The following complications are dealt with:—

*Haemorrhage*, generally easily controlled by packing.

*Escape of bile* occurring a few days after the operation is in the author's opinion not of serious import.

*Pneumothorax* due to opening a nonadherent pleura is not as a rule attended with serious symptoms.

*Parotitis*.—Two cases are described but unfortunately examination of the pus in one of these, which went on to suppuration, was not made, so that it is not known whether this was due to the amoebae.

*Post-operation pleurisy*, of a very insidious type.

*Subphrenic abscess* with usual symptoms.

Part 5. Prognosis.—

The mortality from liver abscess in Tonkin is 50 per cent. and this high figure is accounted for by the acute type of the disease frequently seen, and by the cachectic condition of many of the patients.

S. R. D.

GAIDE. *Le Diagnostic Différentiel des Kystes Hydatiques et des Abscesses du Foie (Considérations cliniques).*—*Ann. d'Hyg. et Méd. Coloniales.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 5-17.

This paper consists of a detailed account of a case of suppurating hydatid cyst of the liver which up to the time of the operation, performed for the evacuation of the pus, was considered to be an abscess of the liver consecutive to dysentery contracted in Madagascar. The case ended fatally. The author ends his paper with a long discussion on the differential diagnosis between amoebic abscess of the liver and hydatid cysts.

S. R. D.

GREY (George M.). *Liver-Abscess.*—*Jl. London School Trop. Med.* 1913. Apr. Vol. 2. Part 2. pp. 141-143.

The report of a case of liver abscess of the very acute type.

On the second attempt exploration by aspiration revealed pus and a Cantlie's trocar and canula were inserted with a view to drainage. The patient, however, died the same night. Post-mortem examination showed that there was a large abscess cavity with ragged walls; no sign of dysentery could be discovered.

Commenting on this case the author points out the unreliability of aspiration and advises laparotomy in its place; he also deprecates the use of Cantlie's tubes except under exceptional circumstances.

S. R. D.

ROUGET. *Abscess Amibien du Foie traité par la Ponction évacuatrice et les Injections sous-cutanées d'Emetine.* Guérison.—*Bull. et Mém. Soc. Méd. des Hôpitaux de Paris.* 1913. Apr. 24. 3 ser. Vol. 29. No. 13. pp. 809-812.

The report of a case of liver abscess occurring in a patient who had suffered from dysentery fourteen years previously when resident in Saigon, during which period he had had several attacks of hepatitis.

The abscess was treated by aspiration and subcutaneous injections of emetine hydrochloride were given in doses of four to six centigrams per diem; the number of injections was six and the total emetine salt administered was 0.28 grams. Recovery was uninterrupted.

S. R. D.



**MALLANNAH (S.). The Value of Emetine in Liver Abscess.** [Memoranda.]—*Brit. Med. Jl.* 1913. June 7. pp. 1206-1207.

The report of a case of liver abscess which had, owing to the patient refusing operation, been treated by repeated aspirations; the pus however quickly recollected and the patient's general condition was rapidly becoming worse. At this stage treatment with emetine was commenced, one quarter of a grain dissolved in distilled water being given three times a day by the mouth. No further aspirations were employed. In spite of this the fever subsided in a week, the liver which was much enlarged subsided to its normal size in one month, and the patient steadily improved. Twenty-one grains of emetine were administered.

S. R. D.

**SEWELL (E. P.). A Case of Amoebic Abscess of the Liver which had burst into the Lung, cured by Emetine Hydrochloride.**—*Jl. R. Army Med. Corps.* 1913. June. Vol. 20. No. 6. pp. 700-702. With 1 chart.

An account of a case of a somewhat chronic liver abscess which had burst into the lung. The patient in spite of surgical interference on two occasions was rapidly becoming worse. Ipecacuanha given in doses of twenty grains a day had no beneficial effect. Emetine hydrochloride was then given (two-third grains subcutaneously daily) and immediately improvement commenced and the patient recovered.

The author also mentions that he has treated cases of chronic amoebic dysentery with emetine salts with uniformly good results.

S. R. D.

**MÜLLER (O.). Die Diagnose und Behandlung des dysenterischen Leberabszesses.** [The Diagnosis and Treatment of Dysenteric Liver-abscess.]—*Arch. f. Schiff- u. Trop.-Hyg.* Vol. 17. 1913. May. No. 9. pp. 289-303; and No. 10. pp. 335-351. With 2 plates and 3 text-figs.

The author gives his experience of liver abscess, consequent upon dysentery, of which he has seen twenty-eight cases in the course of twelve years' residence in Hong Kong. In opposition to the usual belief, he does not find that indulgence in alcohol, in hot climates, predisposes in any great measure to this disease. Only one of his patients could be called a heavy drinker, while several from their position in life, such as missionaries, were practically total abstainers. Bad nourishment on a badly-cooked and scanty diet would seem to be a much more potent cause of the complaint, married men with comfortable homes being relatively exempt. Among the author's patients there was not a single woman, though European women in Hong-Kong are nearly as numerous as the men. Public prostitutes, though often heavy drinkers, for this reason seem to escape. Chinamen also seem immune. Though the author has a large practice among Chinese, he has met with only one case of liver-abscess amongst them. One of his patients was a Japanese, one a Eurasian and one a

Portuguese half-caste, the rest being Europeans, and for this reason the author thinks that there is some kind of racial and sexual proclivity to this complication of dysentery.

Liver abscess, when due to dysentery, is very likely to escape recognition by the European practitioner of medicine, when new to the tropics. In German text-books of medicine the treatment of the subject is undoubtedly insufficient; and the inexperienced medical man is likely to put the condition down to malaria, owing to the chronic fever and the debilitated state of the patient. The antecedent dysenteric symptoms may be very trifling in amount, and should therefore always be carefully looked for. When the abscess is deeply situated, diagnosis is undoubtedly difficult. The patient should be stripped and examined on his back, and all regions where pain is complained of should be carefully palpated, including the intercostal spaces. A tape-measure will often show a considerable difference in the girth of the two sides of the chest, and enlarged superficial veins are also a valuable diagnostic sign. The urine should invariably be tested for urobilin, preferably by means of Schlesinger's test (zinc acetate, ten per cent., in alcohol). If the procedure seems to be indicated, the liver should next be probed in all directions, under an anaesthetic, with an aspirator-needle beginning with the right lobe. The freedom of movement of the needle when the abscess is entered is very characteristic.

When the abscess is found the author prefers to evacuate it by an operation in which portions about two inches in length of the eighth and ninth ribs are removed, with the soft parts, so as to expose the pleura. This is then incised and a corresponding incision made into the diaphragm. The inner edge of the incision in the latter is then fixed, by a continuous suture, to the costal pleura so as to shut off the pleural sac, and the liver is then punctured in the track of the aspirator-needle with a large trocar and cannula having a diameter of one centimetre. The cannula is left in situ, and the abscess is thus drained without any attempt being made to scrape its walls, which may cause haemorrhage. After five days the cannula is replaced by a rubber drainage-tube. This procedure is modified as circumstances require. Convalescence should occupy from three to four weeks, and if improvement does not at once set in, the presence of a second abscess should always be suspected. Of twenty-four cases thus submitted to operation six, or twenty-five per cent., succumbed from various complications. The author terminates his paper with a summary of the clinical history of each case.

S. R. D.

LECOMTE. *Carie Costale consécutive aux Abscès du Foie.*—*Ann. d'Hyg. et Méd. Coloniales.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 17-48.

The author reports six cases of necrosis of the ribs consecutive to operations performed for the opening of liver abscesses. The necrosis in all these cases was extensive and required in many cases repeated surgical interference.

The author's conclusions are as follows:—

1. This complication of tropical liver abscess is not very uncommon.
2. It is more frequent in those cases where the abscess has been opened without resecting a rib than in those where an extensive resection has been carried out.
3. It is caused by insufficient drainage, due to too small an opening being made.
4. The necrosis affects usually the anterior part of the ribs and their cartilages and spreads by means of the mammary lymphatics.
5. The complication is difficult to cure and subject to relapses.
6. The best means of prevention is good drainage by means of a large opening both through the superficial structures and the liver and an extensive resection of a rib.

S. R. D.

#### BACILLARY DYSENTERY.

KRONTOWSKI (A.). Zur Frage über die Typhus- und Dysenterie-  
verbreitung durch Fliegen. [The Dissemination of Enteric  
and Dysentery by Flies.]—*Centralbl. f. Bakt.* 1. Abt.,  
Orig. 1913. Apr. 23. Vol. 68. No. 7. pp. 586-590.

According to CAO, the larvae of flies, if infected with bacteria, will retain them alive sufficiently long for the infection to be propagated by the winged insect. GALLI-VALERIO regards this question as an important one in regard to the recrudescence of epidemics. Experiments were undertaken to test this view.

Larvae of several species of fly, *Musca domestica*, *Lucilia Caesar*, and *Sarcophaga carnaria* and *mortuorum*, were enclosed in glass vessels having a layer of earth at the bottom. They were then fed on chopped meat moistened with a broth-culture of *B. typhosus* or *B. dysenteriae* Shiga-Kruse. After the lapse of three days the larvae were transferred to fresh vessels containing earth. In a proportion of the experiments the larvae were washed for one minute in a solution of sublimate (1:1000) to destroy all bacilli adhering to the surface of their bodies. As the flies hatched out, sterile slips of paper were placed in the jars for them to defaecate upon. Cultures were also made from the intestinal canal upon solid media, and in addition flies were placed in contact with sterile milk. The result of all these experiments was quite negative, from which the author concludes that the Galli-Valerio hypothesis is not proved.

The question as to how long dysentery bacilli can remain alive in the bodies of adult flies was next investigated. From twenty to thirty house flies were placed in a glass jar containing bread moistened with a twenty-four hour broth-culture of *B. Shiga-Kruse*. The flies were then isolated in other vessels, and after the lapse of varying periods, cultures were made from them. The flies having been killed with ether-vapour, the wings and legs were cut off, and the bodies were sterilized with formalin-vapour for from ten to forty-five minutes before being opened, control experiments with other bacteria having shown that three minutes contact with formalin-vapour in this way suffices for the purpose of disinfection, while bacteria in the alimentary canal remain active after

forty-five minutes. The general results of cultures made in this way was that the dysentery-bacilli could be isolated for from two to three days after absorption at the outside; all cultivations made from the fourth day onwards gave no colonies of these organisms.

S. R. D.

HUTT. *Neue Beiträge zur Kenntnis der Pseudodysenterie und Paradyenterie, sowie der sogenannten Mutation.* [A Fresh Contribution to our Knowledge of Pseudo- and Paradyenterie, and their so-called Mutation.]—*Zeitschr. f. Hyg. u. Infectiönskr.* 1913. Apr. 25. Vol. 74. No. 1. pp. 108-137.

This is a long and rather inconclusive paper representing a great deal of laborious work which cannot be conveniently abstracted, as it is embodied in a number of elaborate tables. The author undertook to test systematically the permanence of about forty different strains of pseudo-dysentery bacilli collected from various sporadic outbreaks of dysentery in Germany. All the types examined differed conclusively from the SHIGA-KRUSE bacillus of dysentery; but the number of races into which they have been subdivided by KRUSE and his pupils, under the letters of the alphabet, seems excessive. The author's conclusion is that all these various strains are referable to one permanent type the *B. pseudo-dysenteriae* (KRUSE) which is clearly distinguishable from the bacillus of true dysentery, *B. dysenteriae* Shiga-Kruse, but the differentiation should not go further. Such types as Flexner, Strong, and Type-Y are not really maintainable, if the sugar-reactions are taken as a criterion. Agglutination-tests permit of a division into strains but out of KRUSE's forty varieties the author selects, on the strength of his own results, only four, namely, A.D.E. and H, as worthy of classification, of which E has the peculiarity of slowly acidifying lactose and curdling milk. Upon these four he bestows the appellation of principal strains (*Hauptrassen*) and to them he adds B.C. and F. as subsidiary strains (*Nebenrassen*). The latter will not originate epidemics, but can be isolated in many cases along with the others. The permanency of such races must still be regarded as doubtful, and it is possible that they sometimes appear as gas-forming coli-form types. A transition from the true *B. dysenteriae* to *pseudo-dysenteriae* could not be obtained in any of the author's experiments.

S. R. D.

#### MIXED DYSENTERY.

GAUDUCHEAU (A.). *Recherches sur les Dysentéries (5<sup>e</sup> Note). Etude de Quelques Actions Défensives contre les Germes Dysentériques.*—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. Apr. Vol. 4. No. 4. pp. 167-177.

A very disjointed paper containing remarks on the following subjects:—

1. The action of various drugs on cultures of amoebae was investigated and the author found that emulsions of creasote (5

per 1,000) killed amoebae in two minutes. *La liqueur de Labarraque* (composition of which is not given), two to five per cent., killed the amoebae in ten minutes. Ipecacuanha (in what form not mentioned), 1 in 50, failed to show any deleterious effect. Bile obtained from monkeys killed and dissolved the amoebae immediately.

2. Criticising COHENY's explanation of the improvement, sometimes seen in cases treated with enemata of sugar solutions, being due to hypotonicity of this solution towards the amoebae, he suggests that in reality the beneficial result may be due to the alteration of the intestinal bacterial flora.

3. Discussing the relationship of bacteria to cultures of amoebae the author suggests that at times the bacteria may nourish the amoeba by breaking down the nutrient media into a substance suitable for their assimilation.

4. Infection of animals with human pathogenic amoebae gave, in the author's experiments, very inconsistent results and from these he suggests that pathogenic amoebae may be specific for the species of animal infected.

5. With regard to the type of *B. dysenteriae* found in Tonkin the author states that it ferments maltose but not mannite and is therefore closely related to, but not identical with *B. dysenteriae* Shiga.

S. R. D.

**MARTINI.** Ueber Ruhr im Deutschen Schutzgebiet Kiautschou und in Schantung. (Congress des Royal Institute of Public Health, Berlin, 1912. Original-Referate). [On Dysentery in the German Protectorate of Kiao-Chau and in Shantung].—Abstract in *Centralbl. f. Bakt.* 1. Abt. Ref. 1912. Oct. 11. Vol. 55. No. 4-5. pp. 109-110.

The dysentery prevailing in the Protectorate is ascribable to the following agents:

1. *Entamoeba histolytica* (Schaudinn).
  2. *Entamoeba tetragena*.
  3. Probably, to *Uronema caudatum*, a ciliate organism.
  4. B. Shiga-Kruse.
  5. B. Flexner.
  6. B. Type Y.
  7. B. Strong.
  8. A number of widely differing bacilli all referable to the class of moderately toxic dysentery bacilli like Nos. 5, 6, and 7.
  9. Possibly to nematode worms.
  10. Probably to mechanical, thermal and other unspecified causes, not referable to the presence of living organisms.
- This multiplicity of causes, already pointed out by OHNO of Tokio in 1905-1906, indicates the need for further investigation.

S. R. D.

CANTLIE (J.). **Collosol Argentum: Its Use in Sprue and Post-Dysenteric Conditions.** — *Jl. Trop. Med. & Hyg.* 1913. Apr. 15. Vol. 16. No. 8. pp. 123-124.

The author gives as his opinion that the bactericidal action of silver has probably been the cause of the frequent use of this metal in domestic goods.

He then states that finding collosol preparations are highly bactericidal and have great power of arresting fermentation, he tried their effect in cases of sprue and post-dysenteric conditions. The first case treated was one of sprue with very abundant frothy stools; doses of one drachm of collosol argentum (Crookes) were given three times a day with the result that the character of the stools was markedly changed; they became of a greenish colour and the frothiness disappeared. After six doses the drug was withheld but the frothy stools did not recur.

His present method of treating such cases is to administer five to sixty minims of the drug in half an ounce of water every morning till the stools become green, together with a rigid meat diet.

S. R. D.

LI (T. S.). **Etiology, Symptomatology, Diagnosis and Differential Diagnosis of Dysentery.** — *China Med. Jl.* 1913. Jan. Vol. 27. No. 1. pp. 20-24.

This paper consists of a summary of the symptoms, &c. of dysentery culled from various text books.

No original work is mentioned nor are any original suggestions made.

S. R. D.

#### CILIATE DYSENTERY.

WALKER (Ernest Linwood). **Quantitative Determination of the Balantidicidal Activity of certain Drugs and Chemicals as a Basis for Treatment of Infections with *Balantidium coli*.** — *Philippine Jl. of Science*, Sect. B., Trop. Med. 1913. Feb. Vol. 8. No. 1. pp. 1-15.

The author in his preliminary remarks states that, in individuals infected with these parasites, it is only at times that the Balantidia can be demonstrated in stools and in consequence of this spontaneous disappearance it is very difficult to judge the efficacy of a given treatment by repeated examination of the faeces. He further states that sooner or later a patient, harbouring Balantidia in the intestine, will develop dysentery which is very apt to prove fatal.

Up to the present various drugs have been used in this disease, but practitioners have disagreed as to their efficacy. With a view to proving the efficiency of the drugs the author tested their power of killing the Balantidia in vitro. The varieties of Balantidia used in these experiments were *B. coli hominis* and *B. coli suis*. The conclusions arrived at were:—

1. Compounds of arsenic and antimony, the aniline dyes, ipecacuanha, emetine, and quinine possess little or no power of killing these protozoa.

2. The salts of the heavy metals, especially mercury and silver, have a very high balantidicidal power.

3. The organic compounds of silver, which are not precipitated by albumin or if precipitated form soluble compounds with an excess of albumin, possess also a high balantidicidal power and the author suggests that these substances would be the most suitable drugs to experiment with clinically owing to their slight toxicity and their power of penetrating the tissues.

Tables are given showing the dilutions in which the various drugs and chemicals tested killed the Balantidia.

S. R. D.

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## HELMINTHIASIS.

## SCHISTOSOMIASIS.

MIYAGAWA (Y.). *Beziehungen zwischen Schistosomiasis japonica und der Dermatitis, unter Berücksichtigung der Methode der Auffindung von Parasiteneiern in den Faeces, und Beiträge zur Kenntnis der Schistosomum-Infektion.* [The Relationship between Japanese Schistosomiasis and Dermatitis, with special reference to the Method of discovering Eggs of the Parasite in Faeces.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. May 23. Vol. 69. No. 3. pp. 132-142.—*Sei-i-Kwai Med. Jl.* 1912. Sept. 10. Vol. 31. No. 9. pp. 228-229.—*Jl. Tokyo Med. Assoc.* Vol. 26. No. 7.

In those regions in which schistosomiasis is common in Japan there is a peculiar form of dermatitis which has been attributed to cutaneous infection by the young schistosome. The author however discards the theory of a causal relation because (a) the dermatitis occurs in regions where there is no schistosomiasis; (b) the histological changes in the skin in the dermatitis are quite different from those seen in skin containing the young worms; no young worms are found in sections of skin showing typical dermatitis; (c) dermatitis is more frequent in those who have no eggs in their faeces than those who have; (d) the author and his assistant suffered from dermatitis in 1912 but up to the present no eggs have appeared in their faeces.

The search for the eggs was made by a modification of TELEMANN'S method: a mixture of equal parts of 50 per cent. HCl and ether is added to the faeces and the whole centrifuged. The eggs are found in the residue and are not so much damaged as in TELEMANN'S method. There is the further advantage that no danger of an explosion is incurred.

The author summarises a series of valuable facts relative to the infective agent of schistosomiasis as follows:—

The younger worms of the *S. japonicum* seem to exist more often in the bed of the brook than in the running water, but the mud does not, apparently, contain a perceptible quantity of the worms. The infection is not observed in swampy regions but seems to be localised in places where there is plenty of water. It is more numerous in brooks than in rice fields, but occurs frequently in freshly watered rice fields. Quinine hydrochlor has no prophylactic action as was formerly supposed. Thickly woven cotton cloth acts as a preventive of infection to a greater or less extent.

R. T. Leiper.

MIYAKAWA (Y.). *On the Nutrition of the Eggs of Schistosomum japonicum and the Changes which they undergo under the Actions of Various Physical and Chemical Agents.*—*Sei-i-Kwai Med. Jl.* 1913. Feb. 10. Vol. 32. No. 2. [Whole No. 372.] pp. 10-11.

Four types of eggs are distinguished in faeces, viz. fine granular, coarse granular, those with young and those with well-developed miracidia. The characteristics of the dead eggs vary and are



probably due to post-mortem changes at various stages in the development of the egg. The most suitable temperature for hatching of the eggs is between  $25^{\circ}$  C. and  $30^{\circ}$  C. as in the months of July and August. Eggs still alive are found after immersion for three and at times four weeks in April and May. In July and August they have been kept only up to nineteen days. In urine they do not survive much longer than one week. After three but not after four weeks' exposure at  $-7^{\circ}$  C. eggs are capable of hatching when placed in water at ordinary temperature. Eggs put into ice die immediately. At  $37^{\circ}$  C. more eggs die than hatch. Direct sun rays are injurious to hatching and to development. Darkness has no inhibitory action. Lack of oxygen and a five per cent. solution of common salt bring development to a standstill. In urine the power of resistance of the eggs is much weakened and the simplest and safest method of destroying them is to mix the faeces with the urine.

R. T. L.

PEAKE (E. C.). **Additional Notes on the Egg of *Schistosomum japonicum*.**—*China Med. Jl.* 1912. Nov. Vol. 26. No. 6. pp. 350-352. With text-figs.

In a previous paper the author had given an illustrated and detailed description of the egg of *Schistosoma japonicum*. The purpose of the present note is to draw attention to the occasional occurrence in faeces of unfertilised ova of this parasite. These contain no miracidium but are filled with granular material. In size they resemble the ova of the ankylostome, being smaller than the fertilised egg. The shell shows a single sharp outline in contrast to the double contour of the fertilised egg. In both fertilised and unfertilised ova the little curved nipple or spine can usually be found towards one pole. The patient from whom the unusual ova were obtained was carried into the hospital *in extremis*. A differential blood-count showed 54 per cent. of polymorphonuclears, 46 per cent. of lymphocytes, no large mononuclears and, strange to say, not a single eosinophile. This absence of eosinophilia is explained as resulting from the breakdown of the patient's resistance to the verminous infection.

R. T. L.

EDGAR (W. Harold). **Yangtze Fever.**—*Statistical Report of Health of Navy for the Year 1911.* pp. 167-169. [1912. London: published by H.M. Stationery Office.]

For several years a peculiar endemic fever has been recognised in the Yangtze valley, chiefly in the neighbourhood of Hankow. This was often associated with urticaria and was called urticarial fever, and has recently been observed in a considerable number of naval officers. Practically all persons infected have bathed in shallow water or waded in marshy ground near the river, the Europeans generally for the purpose of snipe shooting. Dogs are frequently infected. The prognosis is better in Europeans than in natives, as they are less likely to suffer from reinfection, but even in Europeans the course is often very protracted. [The

disease is held by some to result from cutaneous infection with *S. japonicum*, but MIYAKAWA (see above) maintains it to be clinically distinct.]

R. T. L.

CONOR (A.) & ARROUX. **La Bilharziose en Tunisie: le Foyer du Djerid.**—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 259-261.

In Tunis bilharziasis seems to be endemic between the 33rd and the 34th degree of latitude. In previous communications Conor has given the results of his inquiries in Gafsa and Nefzoua. The authors now show that this affection is sporadic in Djerid. The Bled ed Djerid, "the country of Palms," is in the southern part of Tunis from Gafsa to the edge of the Sahara in the isthmus which separates the Chott Djerid from Chott Rharsa. The inhabitants are chiefly employed in the cultivation of the palms. In the Oases there are numerous springs some of them thermal, and these, especially in El Hamma, are much frequented for bathing by the inhabitants.

R. T. L.

KAY (James A.). **On the Development of the Bilharzia Embryo.**—*Transvaal Med. Jl.* 1913. Mar. Vol. 8. No. 8. pp. 199-205. With 2 plates.

Dr. Kay contributes to the Bilharzia problem some new and remarkable views based upon experiments conducted in Pretoria. He states that he has succeeded in keeping the embryos of *Schistosoma haematobium* alive for 22 days and has followed a series of developmental changes which result in the production of a large number of wormlike organisms.

The miracidium after escaping from the shell only lives a short time even in pure water, its mission being to discharge the germ cells or eggs which eventually develop into an embryo; this new embryo is a small ovoid body about  $\frac{1}{50}$  inch in length. About ten weeks are required for the embryo to arrive at "the mature globular mass of vacuoles ripe for fissuration." This ends the "fifth stage" and the shedding of the vacuoles or breaking up of the embryo ends its direct life. Later there is a development of cells with the formation of two nuclei and one vacuole. The author is unable to trace further stages in his cultures but believes that the nuclei become the mature male and female Bilharzia. He has given a baboon water to drink containing embryos "in the first stage of development" and on the fiftieth day afterwards made a post-mortem examination. No worms or eggs were found in the bladder but in all of several slides of blood and serum "not examined until some days after" Bilharzia embryos were found. The author [wisely] invites other workers to repeat his experiments and is willing to supply cultivations of the embryos.

R. T. L.

ODHNER (T.). Zum natürlichen System der digenen Trematoden, V. Die Phylogenie des *Bilharzia*-Typus.—*Zoologischer Anzeiger*. 1912. Dec. 10. Vol. 41. No. 2. pp. 54-71. With 7 figs.

In this exceedingly valuable systematic paper ODHNER revises the family Bilharziidae (*Schistosomidae* auct.) which now includes the genera *Bilharziella* Looss 1899, *Gigantobilharzia* Odhner 1910, *Ornithobilharzia* (n.g.) Odhner 1912 and *Bilharzia* Cobb. 1859. The author traces the phyletic development of these unisexual blood worms from a hermaphroditic intestinal form resembling *Liolope*. The stages are represented graphically in series thus:—*Liolope*—*Hapalotrema*—*Bilharziella*—*Ornithobilharzia*—*Bilharzia*, and the thesis is supported by a large number of morphological details.

R. T. L.

#### DISTOMIASIS.

POUMEYRAC (M.). Fièvre Bilieuse Hémoglobininurique déterminée par la Distomatose Hépatique.—*Ann. d'Hyg. et Méd. Colon*. 1912. July-Aug.-Sept. Vol. 15. No. 3. pp. 612-613.

The clinical history is given of a patient, admitted to hospital as suffering from malaria, in whom there was a fairly intense and generalised jaundice and vomiting. For several days previous there had been fever in the evenings and a slight degree of jaundice had been noticeable for several weeks.

Quite a distinct haemoglobinuria was observed on the day after admission; on the following morning however the fever had disappeared, and the urine was more abundant and not so dark. In the afternoon the fever returned (at 2 p.m. it was 39.1° C.) and the patient complained of a sharp scapular pain in the right side.

In a similar case observed in the preceding year at Phuhang Thuong a vast number of flukes were found in the liver at the post-mortem examination and in this case too the liver was eventually found to be literally crammed with these parasites. The other organs were healthy save only for some slight congestion in the right kidney.

R. T. L.

THIBAULT. Un Nouveau Cas d'Infestation par *Clonorchis sinensis* chez un Européen au Tonkin.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*. 1913. Jan. Vol. 4. No. 1. pp. 35-38.

The only known cases of infection of a European with the Asiatic liver fluke is that noted by Dr. AUDIAU in 1910 and ROUX and TARDIEU in 1912. This paper adds an additional record, likewise from Tonkin. The *Clonorchis sinensis* has been shown by SÉGUIN, MOUZELS, MATHIS and LÉGER to occur in 30 per cent. of the natives around Tonkin.

In the case of Drs. ROUX and TARDIEU (see this *Bulletin*, Vol. I., p. 430) as many as 15 to 20 eggs of *Clonorchis sinensis* were seen microscopically in each coverslip preparation of the faeces in association with sparse cysts of amoeba and trichocephalus ova. But for this microscopical determination the case

would probably have been overlooked as during the six weeks spent under observation in hospital no symptom of hepatic insufficiency was apparent.

In Thibault's case the number of ova seen in a microscopical preparation averaged four or five. There was no eosinophilia and no history of hepatic insufficiency.

In the discussion following upon the reading of this paper LE ROY DES BARRES mentioned a further case in his surgical practice and DUVIGNEAU quoted two instances of infection of man in Tonkin with *Fasciola hepatica*.

R. T. L.

#### TAENIASIS.

BETTENCOURT (A.). *Sur la Fréquence relative du Taenia solium et du Taenia saginata en Portugal.*—*Arquivos Inst. Bact. Camara Pestana*. 1913. Apr. Vol. 4. No. 1. pp. 1-5.

Of 65 tapeworms received from colleagues in Lisbon and from the provinces 41 were specimens of *Taenia solium* and 24 of *Taenia saginata*, giving for *T. solium* a proportion of 63 per cent., which is much higher than in any other country for which statistics have been compiled. *Bothriocephalus latus* would appear to be entirely absent from Portugal.

R. T. L.

CARINI (A.) & MASTRANGIOLI (F.). *L'Hymenolepis nana nello Stato di S. Paulo (Brasile).*—*Giorn. d. R. Accad. di Med. di Torino*. 1912. Oct.-Nov. Vol. 75. No. 10-11. pp. 297-300.

Since 1908 the authors have observed the eggs of *H. nana* in eleven cases, of whom four were adults, the others boys from six to fifteen years of age. In one of these cases which showed a large number of ova in the stool the administration of a vermifuge resulted in the removal of about 200 worms. The following symptoms are attributed to this infection:—pallor of the skin and mucous membranes, abdominal pains, diarrhoea, and nervous excitability.

R. T. L.

#### ANKYLOSTOMIASIS.

MALVOZ (E.). *Dix Années de Lutte contre l'Ankylostomiasie des Mineurs.*—*Bull. Acad. Royale de Méd. de Belgique*. 1913. Mar. 29. Ser. 4. Vol. 27. No. 3. pp. 264-278.

The ankylostomiasis, which was formerly a source of much invaliding amongst the workers in the mines around Liège, has in the course of ten years been brought under complete control. The campaign consisted essentially in the tracking of all worm carriers whether sick or not, and their subsequent treatment under efficient supervision. During the course of treatment instruction in the principles of personal prophylaxis was given to the patients. The stools of all new comers and periodically of the whole personnel of the mines were microscopically examined. The result has been that an average of 23 per cent. of worm

carriers in 1902 has dwindled to less than two per cent. in 1912. Further the carriers can now be determined only by microscopical examination. There are no more patients!

R. T. L.

- i. FERRELL (Jno. A.). **Methods for the Eradication of Hookworm Disease.**—*Amer. Jl. Public Health.* 1913. May. Vol. 3. No. 5. pp. 492-493.
- ii. ROCKFELLER SANITARY COMMISSION. **Third Annual Report of the Rockefeller Sanitary Commission for the Eradication of Hookworm Disease.**—130 pp. With 42 illustrations. 1912. Washington, D.C., U.S.A., Offices of the Commission.
- iii. STILES (Ch. Wardell). **Soil Pollution. The Chain Gang as a Possible Disseminator of Intestinal Parasites and Infections.**—*U.S. Public Health Rep.* 1913. May 23. Vol. 28. No. 21. pp. 985-986.

These three publications give an account, the first mainly popular, the latter two official, statistic and scientific, of the vast anti-Hookworm Campaign that is now being waged in the United States of America under the financial aegis of Mr. ROCKFELLER. It is believed that the eradication of hookworm disease is dependent upon the installation and uniform use of sanitary privies by all the people in infected areas and the successful treatment of every infected person. The wholesale adoption of these simple remedies depends in a civilised community upon the proper education of the people as to their practical effectiveness and this is most convincingly demonstrated by the curing of known sufferers of the disease.

Everything is done to induce the general populace to be examined and treated. [The methods remind one forcibly of those adopted in this country by politicians and by the financial organisers of religious and charitable causes.] There are State and County dispensaries for the free examination and treatment of the disease for all applicants. The active co-operation of the doctors, teachers, editors and the leading citizens is obtained by personal visit or letter. In addition the country is being flooded with newspaper announcements, posters and handbills giving information relative to the itinerary, the bringing in of specimens of faeces, etc. The crowd assembled on the first dispensary day witnesses the use of the microscope, the collection of intestinal parasites, an exhibit of photographs of cured and uncured persons, charts, literature, etc., and much interest is aroused from the beginning. Each successive week finds the crowds increasing in size bringing back again and again those infected until cures are made.

The results of treatment in many cases are marvellous. It is no uncommon thing to find that there is a gain in weight of a pound a day. "Patients brought in on stretchers have been restored to normal health in a few weeks at a cost of only a few cents. The witnessing by the people of the transformation from invalidism, blighted ambition, misery and poverty to health, happiness and productive activity does the teaching which excites them to action."

The total number of persons recorded in the annual report for 1912 as having been treated in eleven States is 238,755, bringing the total for the three years during which the Commission has been working to 393,556 at an average cost per person of \$1.02.

During this period 432,464 microscopical examinations were made and inquiries conducted to determine the degree of infection in country school children and the degree of soil pollution at farmhouses, etc.

The report is illustrated by a large series of illustrations showing the effects of treatment and the methods adopted to popularise the campaign; and concludes with a valuable report by the Scientific Secretary Professor Stiles. Three points in this are especially worthy of note.

(a) Hookworm disease is one of the commonest causes of amenorrhea and irregularity, especially of the delayed type and is very frequently overlooked as the exciting cause.

(b) The standard routine of treatment is as follows:—

*First Day* 6 or 8 p.m. Epsom Salts.

*Second Day* 6 a.m.  $\frac{1}{2}$  of total dose of Thymol.

8 a.m.  $\frac{1}{4}$  " " "

No breakfast.

10 a.m. Epsom Salts.

Noon, light lunch.

Stiles is led to modify this routine thus:—

(a) First day 5 p.m. Epsom Salts followed by copious drinks of water.

6 p.m. light supper.

8 p.m. Epsom Salts, followed by copious drinks of water.

(b) Second day 6 a.m.  $\frac{1}{3}$  total dose of Thymol.

7 a.m.  $\frac{1}{4}$  " " "

8 a.m.  $\frac{1}{4}$  " " "

By the three-dose method the same number of grains is taken into the intestines in the same number of hours. Where the patient shows some idiosyncrasy to thymol or when symptoms of thymol poisoning develop, the three-dose method permits the idiosyncrasy or symptoms to be discovered more promptly and at a time when a smaller dose is in the system.

From a considerable amount of chemical work it is concluded that less than ten per cent. of the administered thymol is excreted unchanged in the faeces. Ninety per cent. of each dose of thymol must therefore either be decomposed or be absorbed from the intestinal tract, a proportion certainly not anticipated on the basis of clinical observations.

In "Soil Pollution" Professor Stiles complains that, save in a few instances, the American authorities not only neglect to utilize the opportunities of inculcating lessons in cleanliness, hygiene, and sanitation amongst prisoners but actually permit and even compel their prisoners to live under conditions of filth ideal for the spread of soil pollution diseases.

R. T. L.

COCKIN (R. P.). *Ankylostomiasis in Grenada.* — *Parasitology*. 1913. Apr. Vol. 6. No. 1. pp. 57-67.

The unusually large number of patients suffering from some degree of dilatation of the heart and the occurrence of an intermittent fever which did not respond to quinine or was explained by the finding of malarial parasites in the blood led Dr. Cockin to conduct a series of investigations into these maladies during his official residence in Grenada.

Of the sick populace sent to the Hospitals for treatment and varying in age from young children of three years to old men of 85, no less than 62·78 per cent. in a series of 1,400 were found to harbour eggs of ankylostomes. Of these only two per cent. actually came for treatment for ankylostomiasis and not more than 45 per cent. showed definite symptoms of this disease.

Examination of the urine showed the presence of albumin in fourteen per cent. of the cases infected with the parasite and this occurred quite as frequently in early as in late cases. The following figures are given to bear out the striking association noted between the occurrence of albuminuria, cardiac dilatation and ankylostome infection.

—	Albuminuria.	Cardiac dilatation.	Albuminuria and cardiac dilatation.
Associated with ankylostomiasis.	52	70	37
Not associated with ankylostomiasis.	9	2	2

Thus the parasite was present in 92 per cent. of the cases, an association out of all proportion to the degree of incidence obtaining amongst the sick population generally. The author concludes that while arduous labour in a hilly country does affect the cardiac condition it does so by aggravating an already diseased organ.

From a series of 25 blood examinations, made in cases in which the disease was advanced, the haemoglobin was estimated at 20·5 per cent. and the differential leucocyte count showed an eosinophilia of 22·4 per cent.

A history of ground-itch was obtained in 93 per cent. of the cases.

The intermittent fever was inconstant and did not depend upon the severity of the infection: the expulsion of the parasites proved the only efficient febrifuge. Constipation was the rule but occasional cases of diarrhoea, associated with the passage of blood and mucus extending over a period of years, recovered after treatment which resulted in the expulsion of a large number of parasites. Dr. Cockin's experience is strongly in favour of Beta Naphthol as a rapid, complete, safe and cheap vermifuge. "Haemoglobinuria" occurred however in one per cent. of the cases treated by this drug. His suggestions as to the measures to be taken to free the Colony of the disease follow upon well-known lines.

R. T. L.

BROWN (B. W.). **Hookworm Disease in Southern China.**—*U.S. Public Health Rep.* 1913. Feb. 7. Vol. 28. No. 6. pp. 250-252.

The immigration authorities of the United States now require a rigid examination for ankylostome infection of all aliens arriving in the United States. The question of the distribution of this disease in the Orient has consequently assumed one of economic importance. The author has collected statistics from Dr. BELL in Hongkong who reports 7·5 per cent. of cases in 253 Chinese, 10·5 of 172 Hindus and 0 per cent. in 159 Europeans examined by him.

Dr. WHYTE at Swatow gives the degree of infection of his district as 74·5 per cent. of the farmers and 54 per cent. of the general population as a result of the examination of 257 cases. Drs. ERONE, AUBREY and Lindsay Wood find in 556 persons leaving Hongkong from Southern China for the United States 65 per cent. of infections. The town and district of Sun Ning, not far from Hongkong, furnished 46 cases out of 102.

R. T. L.

SCHÜFFNER (W.) & VERVOORT (H.). **Das Oleum chenopodii gegen Ankylostomiasis und eine neue Methode der Wertbestimmung von Wurmmitteln.** [The Action of Oleum chenopodii in Ankylostomiasis and a New Method of evaluating Vermifuges.]—*München. Med. Wochenschr.* 1913. Jan. 21. Vol. 60. No. 3. pp. 129-131.

\*Although it is rather expensive Oleum chenopodii is a highly efficient vermifuge in comparison with Thymol, Eucalyptus, and Naphthol, as shown by the results given in this paper.

The authors suggest that the action of these anthelmintics may be most satisfactorily apprised by comparing the proportion of worms removed in two successive courses of treatment with Thymol with the proportions obtained by substituting another drug alternately in the first and second courses.

They tabulate their cases as follows:—

No. of Patients.	1st treatment.			2nd treatment.		
	Vermifuge.	Ankylostomes.	Ascaris.	Vermifuge.	Ankylostomes.	Ascaris.
153	Thymol ...	3706	434	Thymol ...	765	127
{ 165	Thymol ...	4808	162	Ol. Eucalypt...	774	8 }
{ 354	Ol. Eucalypt...	4315	39	Thymol ...	7116	321 }
{ 309	Thymol ...	8449	652	Naphthol ...	1147	15 }
{ 188	Naphthol ...	3462	70	Thymol ...	1641	316 }
{ 142	Thymol ...	4154	155	Ol. Chenopodii	751	45 }
{ 146	Ol. Chenopodii	8022	244	Thymol ...	820	37 }

\* An account of this paper, on different lines, was published in this *Bulletin*, Vol. 1, p. 700.



These figures give the following percentages for Ankylostomes removed :—

Activity of Thymol	:	Thymol	=	83:17
„ „ „	:	Eucalyptus	=	86:17
„ „ „	:	Naphthol	=	88:12
„ „ „	:	Ol. Chenopodii	=	85:15
„ „ Eucalyptus	:	Thymol	=	38:62
„ „ Naphthol	:	Thymol	=	68:32
„ „ Ol. Chenopodii	:	Thymol	=	91: 9

R. T. L.

CALHOUN (F. P.). **Eye Complications caused by Hookworm Disease with Special Reference to the Formation of Cataracts.**—*Jl. Amer. Med. Assoc.* 1912. Sept. 21. Vol. 59. No. 12. Pt. 2. pp. 1075-1078.

The association of eye complications with hookworm disease has been known for many years. NIEDEN estimated that of 180 cases fully seven to eight per cent. had some retinal changes such as anaemia, arterial pulsation, serpentine veins, great pallor of the disks, and haemorrhages. All of these might occur in chlorosis or other anaemia; nevertheless, characteristic retinal haemorrhages more numerous and earlier in the periphery than near the papilla or macula and massed in groups of punctate form, are typically associated with hookworm disease. Other changes of a more general character are summarised from the literature by ASHFORD as follows:—blurred vision, dilated pupils, diplopia, unilateral and bilateral nystagmus, amaurosis, restriction in visual field, night blindness and other asthenopic symptoms which might occur in any protracted illness with anaemia.

Night blindness is variously estimated as occurring in from three to twenty-four per cent. The condition of the pupil is regarded by STILES as of diagnostic importance:—“If the patient is directed to stare intently into the observer's eyes the pupils dilate, then the patient's eyes assume a dull, blank, almost fishlike or cadaveric stare.”

In the present paper special attention is directed to cataracts in the young and spontaneous haemorrhages from the conjunctiva associated with, if not caused by hookworm infection. Three cases are reported in detail showing the onset of cataract due to the anaemia and toxæmia produced by the hookworms. A remarkable case of spontaneous bleeding from dilated veins in the retrotarsal fold of the conjunctiva in a well-developed girl of eleven years of age is held to be associated with the presence of many ankylostomes in the intestine. The haemorrhages ceased after a course of anthelmintic treatment, an iron tonic and the local application of a weak epinephrin eye-wash. The eye changes are held by the author to be actually due to, not merely associated with, the hookworm infection. Retinal haemorrhages, enlarged veins, and optic neuritis strongly point to a toxæmia producing a degeneration in the fundus and acting upon the capsular epithelium with a consequent cloudiness of the lens.

R. T. L.

STITT (E. R.). **A Quick Method for accurately differentiating the Species of Hookworm of Man.**—*Jl. Amer. Med. Assoc.* 1912. Nov. 9. Vol. 59. No. 19. pp. 1706-1707.

The author believes that the most satisfactory method of demonstrating to students the difference between *Ankylostoma duodenale* and *Necator americanus* is by showing the characteristics of the buccal capsule of each species. This however is extremely difficult unless the worm is so orientated that a direct view may be had of the opening of the mouth, and this can be best be attained by placing the fresh worm under a coverglass in salt solution and causing it to roll by gentle manipulation of the glass with a toothpick. Next attention should be directed to the copulatory bursa of the male. In *N. americanus* a deep cleft separates the two branches of the dorsal ray and they have bipartite lips. In *A. duodenale* this cleft is shallow and the tips of the branches tridigitate. The dorsal ray is not the conspicuous structure usually represented in text books. Another point to which attention is drawn is in the termination of the spicules. In *A. duodenale* the spicules are simple and end as separate hair-like processes. In *Necator americanus* the two spicules are said to fuse at the terminal endings and to possess but a *single* barb like a fish hook.

Lactophenol solution has been found a satisfactory clearing reagent for ankylostomes and even such large worms as *Ascaris lumbricoides*; it can also be used with good result for small trematodes. There is only slight shrinking. The formula is:—

Lactic acid	...	...	...	1 part.
Phenol (cryst.)	...	...	...	1 part.
Glycerin	...	...	...	2 parts.
Water	...	...	...	1 part.

The specimen should be fixed previously in 2 per cent. solution of formalin for from two to twelve hours according to size, then transferred to equal parts of lactophenol solution and water and finally to full strength lactophenol. In this they may be kept indefinitely in a bottle or as permanent mounts on a glass slide if the coverglass is ringed with gold-size.

R. T. L.

SCHÜFFNER (W.). **Notiz über den Bau der Schwanzspitze bei Ankylostomum- und Necator[sic]-weibchen.** [The Morphology of the Tail in Ankylostoma and Necator Females.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1912. Oct. Vol. 16. No. 20. pp. 700-703.

The author discusses the constancy and specific significance of a small spine protruding from the posterior end of the female of *A. duodenale*. He notes its absence in *Necator americanus*. In the females of both species a pair of "canals" can be made out just anterior to the tip of the tail and apparently perforating the cuticle. In Deli (Sumatra) both species come under observation. The *Necator americanus* chiefly from Java, the *Ancylostoma duodenale* from the southern provinces of China.

[The "canals" appear from the illustrations to be minute lateral caudal papillae. It should be noted that in the photograph showing a series of the two different species of ankylostomes in man those exhibiting characteristics of *Necator* are labelled *Ankylostomes* and vice-versa.]

R. T. L.

LANE (Clayton). *Agchylostoma ceylanicum*, a New Human Parasite.—*Indian Med. Gaz.* 1913. June. Vol. 48. No. 6. pp. 217-218. With 1 plate.

A batch of thirty prisoners from Mymensing Jail were treated with MANSON'S mixture for ankylostome infection and the worms were picked out from the faeces after washing and sedimentation. In three of the cases some of the worms were noticeably thinner and shorter than adult specimens of *A. duodenale*. These proved on microscopical examination to belong to the species *A. ceylanicum*, hitherto known only as an intestinal parasite of the civet cat in Ceylon. Its appearance in man is apparently merely sporadic but Lane shows that it commonly infests the cats and dogs in Berhampore, Bengal. An African lion born in the Calcutta Zoo was also found to be infected with this species.

Whereas the type specimens from the civet cat measure on an average 5 mm. in males and 7 mm. in females, in the ordinary cat these figures are 6·8 mm. and 7 mm., in the dog 7·2 mm. and 9·8 mm. and in man (passed after eucalyptus mixture) 8·5 mm. and 10·5 mm. The chief specific characters are given thus—"Two pairs of ventral marginal teeth; when the mouth is looked into from without the one pair is deep and cephalad, and the other superficial and caudad. The points of both pairs are directed caudad. The bursa of the male has marked clefts dividing the dorsal from the lateral lobes; the dorsal ray bifurcates, and each branch again bifurcates, while the edge of the dorsal lobe of the bursa has a single convex curve on each side of the mid-line the two curves producing an outline like that of a weak figure 3. The lateral lobe is rather long and rounded."

Lane points out that should this parasite be shown to be of any clinical significance in man the prophylaxis of ankylostomiasis in India becomes unexpectedly complicated.

R. T. L.

#### OTHER NEMATODE INFECTIONS.

LANE (Clayton). *Trichostrongylus colubriiformis* (Giles 1892), a Human Parasite.—*Indian Med. Gaz.* 1913. Apr. Vol. 48. No. 4. pp. 129-132. With 14 figs.

An opportunity recently presented itself to the author to examine the original material of GILES. From a comparison with the recent descriptions of *Trichostrongylus instabilis* confirmation was obtained for the view that these two species are really identical. The only difference which Lane is unable to explain as due to error in the original description of *S. colubriiformis* is the constantly larger size of the Indian specimens. A

visit to Sanawar enabled the author to determine that the sheep there were infected with this species and to obtain further material for investigation from the "type" locality. The differences in size are attributed "either to racial differences inherent in worms inhabiting hosts in different parts of the world or to difference in methods of preservation." A number of specimens of *Ancylostoma duodenale*, *Necator americanus* and *Oxyuris vermicularis* were afterwards measured and found to exceed on an average the mean given for these worms in other parts of the world. [A portion of the original material was sent to the British Museum by GILES in 1912 and independent confirmation of the above results was published in the Report of the Advisory Committee for the Tropical Diseases Research Fund for 1912.]

R. T. L.

BRAU (P.). *De l'Anguillula intestinalis en Cochinchine et de son Diagnostic hématologique.*—*Bull. Soc. Path. Exot.* 1913. Apr. Vol. 6. No. 4. pp. 262-264.

For some time past the author has remarked in his daily blood examinations at the Laboratory of the Military Hospital in Saïgon a certain degree of eosinophilia, up to 76.34 per cent. even, which was not explainable either by the existence of clinical symptoms or by the occurrence in the stools of the eggs of parasites or even amoebae.

In five recent cases in which the minimum eosinophilia observed was 25 per cent., the maximum 69 per cent., a repeated examination of the stools revealed the presence of *Strongyloides intestinalis*, and the author is of opinion that not only may this parasite have been the cause of this profound modification of the normal blood count but it may well give rise to more serious troubles than those hitherto imputed to it. Progressive anaemia and gastro-intestinal dyspepsia were noticeable in all these cases and in some a chronic diarrhoea of several years duration.

Energetic treatment with thymol resulted, if not in a total elimination of the parasites, at least in a very distinct amelioration of the various symptoms and a noteworthy arrest of the progressive emaciation.

R. T. L.

STILES (C. W.). [Treatment of *Strongyloides* Infection.]—*Science*. 1913. Jan. 31. (new ser.) Vol. 37. No. 944. p. 198.

A brief record is made in some notes on technique and treatment, published in a report of the proceedings of the Helminthological Society of Washington, to the effect that "judging from several cases in which it has been tried, the use of flowers of sulphur seems to be of promise in the treatment of infections with *Strongyloides stercoralis*."

R. T. L.

**RANSOM (B. H.).** *The Life History of Habronema muscae (Carter), a Parasite of the Horse transmitted by the House Fly.*—U.S. Dept. of Agriculture, Bureau of Animal Industry, Bulletin 163. 1913. Apr. 3. 36 pp. With 41 text-figs.

More than fifty years ago CARTER discovered in Bombay that the house fly harboured larval nematode worms to which he gave the name *Filaria muscae*. Their life history remained a mystery and they were regarded of no significance from the public health point of view until Ransom published his preliminary note of the present paper. These recent investigations have proved that these larvae are a stage in the development of a parasite of the horse. The eggs passed in the faeces of the horse are taken up by the fly larvae and shortly before or after the transformation of the flies into the imaginal state the worms reach their final larval stage and reach their maturity when the flies they infest are swallowed by horses. In the flies the parasites are commonly found in the head, frequently in the proboscis, but occasionally also in the thorax and abdomen. The occurrence of these worms in a fly is positive proof that the fly was bred in horse manure; hence the frequency of their occurrence roughly indicates the extent to which horse manure is serving as a breeding place for flies in a neighbourhood.

R. T. L.

**HALL (M. C.).** *A Spurious Parasite reported as Trichinella.*—*Science*. 1913. Jan. 31. (new ser.) Vol. 37. No. 944. pp. 197-198.

In 1908 STACUBLI stated that it would probably be possible to diagnose trichinosis in suspected human cases by examining blood from a finger or ear instead of excising a portion of muscle. The method suggested consists in the centrifugation of the blood after the addition of three per cent. of acetic acid to dissolve the erythrocytes. The embryos would be found in the deposit. Since then three cases have been discovered by this method. HALL considers that the records of HERRICK, JANEWAY (1909) and MERCUR and BARACH (1910) should be accepted: That of CROSS (1910) however is thought to be spurious, there are several indications "that CROSS was dealing with plant hairs or some similar foreign bodies simulating *Trichinella*; the only clinical symptoms given—facial oedema and a forty-four per cent. eosinophilia—leave the case open as far as the existence of a trichinosis is concerned."

R. T. L.

#### GENERAL AND UNCLASSIFIED.

**JOHNSON (W. B.).** *Report on Entozoa Infection amongst Prisoners [in the Zungeru Gaol, Northern Nigeria].*—Received in Colonial Office, May 7, 1913. [Proof.]

Owing to the degree of debility and emaciation met with amongst prisoners in the Gaol at Zungeru, notwithstanding a quite generous and sufficiently varied diet, a microscopical examination was instituted of the stools of the convicts until the total

examined reached two hundred. Evidence of Entozoal infection was obtained in 53·5 per cent. Ankylostome infection was detected in 84 cases (42 per cent.), *Taenia* in 23 cases (11·5 per cent.), *Ascaris lumbricoides* in nine, *Trichocephalus trichiura* in nine, *Oesophagostomum* in eight, *Oxyuris vermicularis* in eight, *Schistosoma haematobium* in eight, and *Strongyloides stercoralis* in five.

The racial incidence of these infections is also tabulated in percentages [it would appear doubtful whether the results based upon so few as seven, and eleven, twelve and fourteen individuals of a tribe could be accepted as supporting the conclusions deduced therefrom].

The author believes that ankylostome infection is the most important cause of the debility so often seen amongst prisoners. He thinks it probable that in the natives' natural state some degree of immunity to the toxæmia is established, but when they are confined to gaol this immunity breaks down under the monotonous although efficient diet, the hard work, and possible mental depression, and the toxæmia becomes a very serious factor. He notes a marked improvement in the general condition after ankylostomes have been expelled in such cases.

In most of the cases under observation malnutrition was the only clinical indication of infection; in five however the disease terminated fatally. The degree of anaemia and emaciation depends not so much upon the number of worms present as upon the duration of infection and of other depressing influences and the presence of other entozoa. Of 35 cases 21 were due to *Ankylostoma duodenale* alone, in the remainder both *A. duodenale* and *Necator americanus* occurred. No instance of a pure infection with *N. americanus* came under observation.

Johuson's experience in treatment coincides with that of other workers. He finds the eucalyptus method inefficient and relies upon thymol, repeated in most cases after four days. In a series of weights before and after treatment for Ankylostomiasis some striking instances are given of the gain in weight, which averaged in 30 cases to no less than twelve and a half pounds. [It is curious however that] the same average gain in weight is shown during the same period by ten prisoners treated for infection with *Taenia saginata* alone.

*Oesophagostomum* in man has been recorded hitherto in two cases only, so that it is of some interest to find eight cases appearing in a systematic examination of 200 men. In only one case was the infection a simple one, in the remaining seven it was complicated with ankylostome infection. In the uncomplicated case no symptoms were noticeable.

R. T. L.

BERNARD (P. Noël) & KOUN (L.). Parasitisme Intestinal en Annam.  
—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5.  
pp. 343-346.

To supplement the published statistics of the Laboratories of Cochinchina and of Tonkin the authors have collated their observations at Hué in Central Annam during the years 1911 and 1912.

The stools of 300 patients under treatment for various, often external affections, and of 149 persons, including 50 mandarins, in perfect health, showed the following incidence of the various intestinal parasites:—

Ascaris ...	...	271 times, i.e.,	60·35 per cent.	
Ankylostomes ...	170	„ „	37·86	„
Trichocephalus ...	87	„ „	19·37	„
Oxyuris ...	10	„ „	2·22	„
Taenia ...	10	„ „	2·22	„
Flukes ...	...	none.		

59·25 per cent. of the cases were infected with one species only. It was not possible to determine the species of ankylostome from an examination of the eggs in the stools. In the occasional post-mortems obtained the *Necator americanus* alone was found. Tapeworms are very common in Hué. Those microscopically examined were *Taenia saginata*. The eggs are only found exceptionally in the faeces for although the statistics given above show only ten positive findings, no less than 66 patients in 1910 and 56 in 1911 presented themselves for treatment. As one per cent. of the pigs killed at the abattoirs in Hué were found infected with *Taenia solium* and two per cent. with *Fasciolopsis rathouisi*, these parasites should not be exceedingly rare in man.

R. T. L.

MAXWELL (James L.). Final Research Report for the Triennial Meeting [of the Medical Missionary Association of China].—*China Med. Jl.* 1913. Mar. Vol. 27. No. 2. pp. 102-104.

In view of the enormous area covered by the membership of the Medical Missionary Association of China and its exceptional facilities for studying parasitic infections a few of the more enterprising members formed a Research Committee some years ago and appealed to the others to undertake certain investigations or to forward material and information. Very useful work was accomplished and it is with much regret that one reads in this the organising "Research Commissioner's Report" that the work is to be discontinued. Surely the exceedingly valuable observations which the Japanese have so frequently reported during the period will stimulate emulation in the workers in the same field in China.

The Commission has been able to contribute to helminthological knowledge at least three important matters (a) The universality of ankylostome infection in China; (b) The widespread occurrence of *Fasciolopsis buski* of slight intensity. In a comparatively small area near the mouth of the Yangtse River the infection is severe; (c) *Schistosoma japonicum* is confined in its distribution to the Yangtse Valley; (d) Dr. HOUGHTON believes that he has found eggs of *Fasciola hepatica* in the stools of several patients.

R. T. L.

SNELL (J. A.). Report of Faeces Examination of 424 Cases in the Surgical Service of the Soochow Hospital. [Final Research Report.]—*China Med. Jl.* 1913. Mar. Vol. 27. No. 2. p. 105.

Since October 1911 the author has made a routine examination of the faeces of patients under his charge as it was apparent that their condition due to the presence of intestinal parasites was often more serious than the particular surgical condition for which they had entered the hospital. The results are tabulated as under. The percentage of ankylostomes is believed to be higher than that shown in the table. *Fasciolopsis buski* is not very abundant in Soochow. Most of the cases come from Kunshan, near Shanghai, and Changshu, about thirty miles to the north of Soochow.

	Cases.	Percentage.
Nematoda :		
<i>Ascaris lumbricoides</i> ...	268	63.2
<i>Trichocephalus trichiura</i> ...	64	15.1
<i>Ankylostoma duodenale</i> ...	41	9.7
<i>Oxyuris vermicularis</i> ...	1	0.23
Trematoda :		
<i>Fasciolopsis buski</i> ...	11	2.6
<i>Schistosoma japonicum</i> ...	4	1.94
Unidentified ova ...	3	0.7

R. T. L.

WOOD (H. B.). Intestinal Parasites in the South.—*Jl. Amer. Med. Assoc.* 1912. Nov. 9. Vol. 59. No. 19. pp. 1707-1708.

The subjoined tables represent, as far as the records are available to the author, the results of the examinations of faeces made in the State laboratories in the Southern States of U.S.A. exclusive of Alabama, Louisiana, South Carolina, and Texas. The figures are those for 1911 and the first quarter of 1912 with some exceptions. The results of the Rockefeller Sanitary Commission are not included.

Table 1.—Summary of the intestinal parasites reported.

Total examinations reported	...	...	62,786
Total showing hookworm infestation	...	...	11,147
Percentage of hookworm infestation...	...	...	17.7
Percentage of infestation by other parasites			6.5
		No. of worms found.	Ratio to other parasites.
<i>Necator americanus</i>	...	11,147	73.13
<i>Ascaris lumbricoides</i>	...	2,339	15.34
<i>Hymenolepis nana</i>	...	1,004	6.59
<i>Trichocephalus dispar</i>	...	551	3.62
<i>Strongylus</i> [ <i>Strongyloides</i> ] <i>intestinalis</i>	...	119	7.9
<i>Oxyuris vermicularis</i>	...	72	4.7
<i>Taenia saginata</i>	...	10	0.6



Table 2.—Ratio of parasites found in various States.

	Arkansas.	Florida.	Georgia.	Kentucky.	Mississippi.	North Carolina.	Tennessee.	Virginia.
<i>Necator americanus</i> ...	94.5	41.6	94.7	40.9	90.9	82.9	47.1	65.7
<i>Ascaris lumbricoides</i> ...	2.7	7.3	1.9	42.8	1.3	17.9	34.4	26.3
<i>Hymenolepis nana</i> ...	1.1	39.6	2.8	0.9	3.7	5.6	5.0	3.1
<i>Trichocephalus dispar</i> ...	0.3	10.6	0.1	15.4	—	3.1	8.6	3.2
<i>Strongylus</i> [ <i>Strongyloides</i> ] <i>intestinalis</i> .	0.7	0.8	—	—	15.0	1.1	0.2	—
<i>Oxyuris vermicularis</i> ...	0.3	1.0	0.2	—	0.4	—	2.6	0.9
<i>Taenia saginata</i> ...	—	—	—	—	0.4	0.03	1.1	—

[The extraordinary frequency of *Hymenolepis nana* is one of the most interesting results of the recent important statistical surveys made in the United States.]

R. T. L.

WARD (H. B.). Means for the Accurate Determination of Human Intestinal Parasites.—*Illinois Med. Jl.* 1912. Oct. 18 pp. With 46 text-figs.

This paper gives a brief and lucid account of the manner in which the chief helminthic infections of man may be accurately detected from a microscopical examination of the faeces. The peculiarities and sizes of the various eggs and embryos are described and beautifully portrayed.

R. T. L.

BEDSON (S. Philipps). Lésions des Organes à Sécrétion interne dans l'Intoxication vermineuse.—*Compt. Rend. Soc. Biol.* 1913. May 16. Vol. 74. No. 17. pp. 994-996.

The suprarenal capsule presents the most important lesions, of all the organs with internal secretion, in acute or chronic intoxication with verminous products. The thyroid reacts in a similar manner, more especially in subacute and chronic but only very slightly in acute intoxications. The hypophysis, pancreas, ovaries and testicles merely show insignificant lesions. The seriousness of the lesions of the suprarenal and thyroid is related rather to the number of injections and the duration of the intoxication than to the amount of toxin injected. The verminous product of ascarides and of some [unidentified] species of taenia produced identical lesions.

The organs of internal secretion react to verminous toxins in the same manner as to microbic toxins or to harmful chemicals.

R. T. L.

WEINBERG (M.) & SÉGUIN (P.). *Recherches sur l'Eosinophile et l'Eosinophilie (Deuxième Note). Explication de l'Abaissement Considérable du Taux de l'Eosinophilie après l'Opération du Kyste Hydatique.*—*Compt. Rend. Soc. Biol.* 1913. May 30. Vol. 74. No. 19. pp. 1096-1098.

It has been noted by several that the eosinophile index is lowered and sometimes to a considerable degree, within a few hours after operation for a hydatid cyst. The authors have experimented to prove that there is a causal relationship between the absorption of a large amount of hydatid fluid liberated in the course of surgical intervention and this diminution in the number of eosinophiles in the circulation.

A number of guineapigs showing a marked eosinophilia have been subcutaneously injected with six to ten cc. of hydatid fluid in such a way as to provoke as far as possible a local eosinophilia over a wide area. A notable diminution in the eosinophilia became apparent within an hour or two after the injection and this became most marked between six and eight hours after the commencement of the experiment. At this time the eosinophilic infiltration of the local lesion has attained its maximum. An intense infiltration of the subcutaneous and intramuscular connective tissue by polynuclear eosinophiles in the region of the injection was determined microscopically. Usually the eosinophiles begin to reappear in the blood on the day following the operation but occasionally, as has been noted in man, the small percentage of eosinophiles continues for two or three days. It has also been demonstrated experimentally that the abrupt disappearance of eosinophiles from the blood, which has been observed to take place in some cases of echinococcosis in man within one or two hours after operation, is explainable by a previous sensitising of the body by hydatid fluid. This increased sensibility in the reaction of the eosinophiles to the presence of toxic substance affords a new argument in favour of the view that these elements play a rôle in the absorption of certain toxic products.

R. T. L.

#### FILARIASIS.

LEIPER (R. T.). *Report of the Helminthologist, London School of Tropical Medicine, for the Half-year ending April 30th, 1913.—Report to the Advisory Committee of the Tropical Diseases Research Fund.* Received in Colonial Office, May 16, 1913. [Proof.]

*Filaria loa*.—The author gives the results of a search for the carrier of *Filaria loa*, by himself, on the West Coast of Africa, during the latter part of 1912 and the beginning of 1913.

No evidence of infection was obtained from a series of mosquitoes of various species fed upon a patient in whose blood very many *Filaria loa* embryos were present. With the same patient *Stomoxys calcitrans*, *Stomoxys nigra*, *Glossina palpalis*, *Cimex rotundatus*, *Pulex irritans*, *Tabanus par*, *T. socialis*, *T. fasciatus*,

*T. secedens*, also gave negative results. In *Haematopota cordigera* and *Hippocentrum trimaculatum* a slight degree of infection was obtained but development was unequal and slow.

In *Chrysops dimidiata* and *Chrysops silacea* a rapid and uniform development took place. No experiments were made with *Chrysops longicornis*, as this species was only obtained once at Forcados, and none of the four flies which were captured could be induced to bite. The experiments were brought to an abrupt close early in January owing to the lack of material.

*Filaria volvulus*.—At Lokoja, Northern Nigeria, Leiper noted no less than thirteen out of 168 prisoners suffering with *Filaria volvulus* tumours. In none of these were embryos found in the blood. Though large numbers of living embryos were injected subcutaneously and intraperitoneally into a monkey, none ever appeared in its blood. The tumours were usually multiple, each containing two or more nodules. The usual site was the crest of the ilium, but tumours were also noted over the sacrum and over the ribs. Specimens of *Stomoxys nigra* and *S. calcitrans* were fed upon the juice of tumours containing many living embryos. Dissections at later dates gave no evidence of the infection of the flies, although embryos were found in the stomach contents. No other biting flies were available for experiment.

*Filaria medinensis*.—At Calabar it was noted that the cases of guinea-worm in the hospital all came from the upper reaches of the Cross River; as a local infection dracontiasis was quite unknown. Difficulty was experienced in finding cyclops. Later, however, some cyclops developed from mud taken from collections of water around Calabar. The author thinks, therefore, that the cyclops had been kept under control in some manner and suggests that the innumerable small fish, belonging apparently to the genus *Haplochilus*, which abounded there, consumed them, as well as the eggs, larvae and pupae of mosquitoes.

A series of experiments showed that these fish were very hardy and adapted themselves readily to confinement in small tanks of water. Their use in wells containing cyclops is suggested. A possible explanation of the peculiar patchy distribution of guinea-worm in tropical countries may lie in the presence of the natural enemies of cyclops, the intermediate host.

G. C. Low.

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## CHOLERA.

ALESSANDRINI (Giulio) & SAMPIETRO (Gaetano). **Sulla Vitalità del Vibrione Colerigeno nel Latte e nelle Mosche.** [The Viability of Cholera in Milk and in Flies.]—*Ann. d'Igiene Sperimentale*. 1912. Vol. 22. (new ser.) No. 4. pp. 623-650.

In 6 experiments with different crude milks inoculated with cholera and kept at room temperature five showed no cholera after 2 days (two not even after one day), but one contained living vibrios up to 63 hours. Similar results were obtained with 4 milks inoculated with cholera stools, *i.e.* the organisms were recoverable up to 62 hours in one case, but in the others not after 2 days. If the samples were kept at 37°, the organisms disappeared in 6-8 hours. In all cases there was first a rapid multiplication, so that after 6-12 hours at room temperature the milk seemed to contain almost a pure culture of the cholera vibrios. Then the numbers fell off, at first relatively to other organisms but later absolutely, and eventually the cholera disappeared almost suddenly. This sudden disappearance is due to the increasing acidity of the milk, and it is to be noted that it is in comparatively fresh and therefore saleable milk that the vibrios are to be found in the greatest numbers. When the acidity reaches a degree corresponding to that of 9·5 or 10 per mille of lactic acid, they die out. In boiled or sterilised milk vibrios remain alive after inoculation up to 60 days when the culture is pure, and even when mixed cultures are added they remain longer than in raw milk.

Flies fed on material containing cholera vibrios in large numbers were found to carry the organisms on their surfaces and in their faeces for 24-36 hours. Similarly larvae could carry cholera on their surfaces, but only rarely were vibrios found in their faeces or intestine. Pupae did not carry the organisms; and flies which developed from larvae that had been kept in faeces full of cholera did not contain nor carry any of the vibrios. The author never recovered cholera from flies caught in conditions of actual practice, where there was reason to believe that contamination might have occurred; but he is prepared to admit that infection of milk may occur in this way.

J. Henderson Smith.

KRUMWIEDE, Junr. (Charles) & PRATT (Josephine S.). **Dahlia-Agar als Unterscheidungsmittel zwischen Cholera- und anderen Vibrionen.** [Dahlia-agar as a Differential Medium for Cholera.] — *Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Apr. 16. Vol. 68. No. 5-6. pp. 562-566.

SIGNORELLI pointed out that, if cholera vibrios be grown on agar to which some dahlia has been added, the developing colonies take up all the colouring matter and appear tinted against the uncoloured background; and he believed that this might prove of service in the differentiation of cholera from other vibrios. Krumwiede and Pratt, however, found it of no real value. The dye must be used very weak (1 in 50,000 or

even less) if it is not to inhibit the growth of the organisms, and no qualitative or quantitative distinction can be drawn between true cholera and other vibrios, either in resistance to the inhibiting action or intensity of staining. It was noticeable that the colonies were less deeply coloured in the strains which grew best.

J. H. S.

STOKES (W. Royal) & HACHTEL (Frank W.). **The Use of a Modified Hesse's Medium for isolating the Typhoid Bacillus and the Cholera Spirillum from Stools.**—*Centralb. f. Bakt.* 1. Abt., Orig. 1913. June 4. Vol. 69. No. 4. pp. 346-349.

The medium for cholera is made as follows. Of agar (dried for half an hour at 105° C.) 5.5 gm. are dissolved in 500 cc. of distilled water. To another 500 cc. are added 5 gm. Liebig's extract of beef, this is filtered and inoculated with *B. coli*; after 48 hours at 37° it is again filtered, and 10 gm. Witte's peptone added and also 8.5 gm. NaCl. This fluid is added to the 500 cc. agar solution, the whole boiled and filtered, 10 gm. starch added, the fluid made neutral to phenolphthalein, and then coloured with Kahlbaum's azolitmin. It is tubed, and autoclaved at 16 lbs. pressure for 20 minutes. In this medium intestinal spirilla give pink colonies; other colonies give blue colonies because they have no amylolytic ferment. The spirilla cannot be distinguished amongst themselves by this medium.

J. H. S.

BAUJEAN. **Etude comparée des Actions protéolytiques et hémolytiques de quelques Vibrions Cholériques.**—*Compt. Rend. Soc. Biol.* 1913. Apr. 25. Vol. 74. No. 14. pp. 799-800.

For estimating the proteolytic power the Chamberland filtrate of 2, 4, 6, or 8 day peptone-water cultures was added to 25 per cent. gelatine, and the time required for liquefaction determined. Ten strains were examined and in all the maximum power had been attained by the 2nd day, and in most had fallen again by the 4th day. The haemolytic power was tested against the corpuscles of rabbit, goat, guinea-pig, horse and man by inoculating tubes containing corpuscles with the vibrios. Some strains haemolysed none of the corpuscles, some laked all the kinds, and some laked certain kinds but not others. No connection was observed between the haemolytic and the proteolytic power—*e.g.* a strongly liquefying strain might have no haemolytic activity.

J. H. S.

BLOEDORN (W. A.). **Report of a Case of Cholera in the U.S.S. "Helena" and Notes on a Shanghai Epidemic.**—*U.S. Naval Med. Bull.* 1913. Apr. Vol. 7. No. 2. pp. 251-252.

A brief note of a case. In the foreign population of Shanghai (14,000) 23 cases of cholera or choleraic diarrhoea occurred during August and September with a mortality of 45 per cent. It is believed that flies played a part in spreading the disease.

J. H. S.

WHYTE (G. Duncan). **The Treatment of an Epidemic of Cholera by Rogers' Method. Based on a Study of 215 Cases which required the Intravenous Infusion of Saline.**—*China Med. Jl.* 1913. Mar. Vol. 27. No. 2. pp. 107-116.

The author refers only to cases in which the blood-pressure fell below 70 mm. mercury, *i.e.* to cases in which intravenous injection was practised. Of these there were 215 with 150 recoveries (70 per cent.); and he discusses chiefly the causes of death in the remaining 65 cases. The three main causes of death were collapse, hyperpyrexia and uraemia. The only reliable indication of impending collapse is a low or rapidly falling blood-pressure, and it can be guarded against only by repeated examination. In cases which develop quickly this must be done frequently, as collapse occurs more rapidly in such cases than in those which follow a slow course; and it is to be kept in mind that sometimes, where the difference between the systolic and diastolic pressures is great (as in aortic incompetence), the usual reading will give a wrong impression of the state of the patient. Vaso-constricting drugs were found of value, and the addition of glucose to the injection is recommended as a food where the patient is rejecting all food by the mouth. To correct hyperpyrexia ROGERS advises injections of reduced temperature, but this, according to Whyte, is effective only if the patient was febrile before the injection, and does not obviate the more frequent cases of fever due to the injection itself. The saline should be filtered, and in children should always be given subcutaneously. Hyperpyrexia accounted for all the deaths which occurred in children and for half the total of 65 deaths. Uraemia should be treated by raising the blood-pressure by saline injections and drugs, at the same time lowering the salt given as much as possible, and finally by cupping over the kidneys to reduce congestion. About 7 per cent. of the patients treated died from this cause.

J. H. S.

NAAME. **Sulla Cura del Colera con l'Adrenalin.** [On the Cure of Cholera by Adrenalin.]—*Gazz. d. Ospedali e. d. Cliniche.* 1913. Apr. 3. Vol. 34. No. 40. p. 415.

PIOVESANA. **Ancora della Cura del Colera.** [Further Remarks on the Cure of Cholera by Adrenalin.]—*Ibid.* Apr. 17. No. 46. pp. 481-482.

Piovesana claims priority in the use of adrenalin in the treatment of cholera as against Naame, who also claims it.

J. H. S.

SCHOPPER (K. J.). **Erfahrungen über die Cholera in Ostrumelien während des Balkankrieges 1912.** [Cholera in Eastern Rumelia during the Balkan War.]—*Wien. Klin. Wochenschr.* 1913. Mar. 6. Vol. 26. No. 10. pp. 366-370.

Four bacteriologists were sent from WEICHSELBAUM's laboratory to Sofia, where they arrived on November 29, *i.e.*, about 14 days after cholera had broken out at the front in the Turkish troops.

In Sofia two cases had occurred before their arrival, but rigorous precautions kept further suspicious cases out of the city in an isolation hospital, where Schopper met with one other case. Numerous journeys were made to different quarters, from which reports of suspected cases were received, and a number of cholera cases recognised. Some of these were amongst the Turkish prisoners, but in every other instance the disease had been brought by transport-drivers. For the most part he found the sick herded together without precautions and under the most insanitary conditions, and it was with the greatest difficulty that even the most elementary measures of isolation could be carried out. The actual number of cholera cases, however, was not very large, and the disease died out almost suddenly towards the end of December. In all, 120 cases of intestinal disease were fully examined bacteriologically, of which 28 were cholera, 2 typhoid, 3 paratyphoid, 8 dysentery (7 being due to Flexner's bacillus). The cholera case mortality was 35·7 per cent.

J. H. S.

SEBASTIANI (Antonio). *Sui Vaccini Colerici*. [Cholera Vaccines.] —*Ann. d'Igiene Sperimentale*. 1912. Vol. 22. (new ser.) No. 4. pp. 569-598.

The author compares the effect produced in guineapigs and rabbits by different vaccines prepared from cholera vibrios. Of these he used one made by KOLLE's method, one according to WRIGHT, a nucleoproteid according to LUSTIG and GALEOTTI, an autolysed emulsion in distilled water and aggressins obtained from guineapigs. [We are unfortunately not given any means of comparing the dose employed of the two last with the others.] All these vaccines rendered guineapigs immune to five lethal doses of culture, but only the aggressins did so after one dose. The aggressins alone produced no marked rise in temperature, but they were not free from harmful results; and they produced the largest quantity of agglutinins in both guineapigs and rabbits. They also gave rise to the largest production of bacteriotropic substances, but all the others were effective in this respect. All the vaccines produced sera which fixed complement, notably the autolysed emulsion. A marked difference was observed in the production of bactericidal substance. Whereas both the Wright and Kolle vaccines produced sera which were strongly bactericidal, the serum obtained after inoculation with nucleoproteid or aggressin was scarcely bactericidal at all.

J. H. S.

CARAPELLE (E.). *Sul Colera del 1910-1911 in Sicilia e Specialmente in Palermo*. [The Cholera of 1910-1911 in Sicily and especially in Palermo.] —*Ann. d'Igiene Sperimentale*. 1912. Vol. 22. (new ser.) No. 3. pp. 451-495. With 14 figs. and 2 maps.

Cholera appeared in Sicily in 1910, imported into Palermo presumably from Naples, and after causing 504 cases or 0·13 per mille of the population, died out again in January 1911. In May

1911 it reappeared, the cause of the recrudescence being obscure, and this time it invaded nearly half the communes of Sicily and caused 7,834 cases or 2.08 per mille. In both years it was most severe in Palermo, whence it spread along the lines of communication through the island. In Palermo itself there were in this second epidemic 3,112 cases with 1,545 deaths. The author discusses the two epidemics on general epidemiological grounds, and concludes that while the 1910 epidemic was perhaps not certainly (at least in Palermo) a water-borne outbreak, that of 1911 was in the main carried by water. In the city the chief source of infection was the drinking water; in the borough-towns in the province, *e.g.*, in Brancaccio, the superficial irrigation water also spread the disease.

J. H. S.

**SALIMBENI (A.) & ORTICONI.** *Essais de Traitement des Porteurs Sains de Vibrion Cholérique par les Lavements de Sérum Spécifique.*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 306-308.

During an epidemic at Marseilles in 1911 34 persons in one Asylum were found to be carrying the vibrios without showing any definite symptoms of cholera, although some had slight choleraic diarrhoea. All these were treated as follows. Each received an evacuating enema and from the faeces so obtained cultures were made. Immediately after this an enema of 50 cc. of cholera serum in 200 cc. of salt solution was slowly injected, attempt being made to let the injection penetrate as far into the intestine as possible. In 9 out of the 34 the vibrios had disappeared at the time of the first injection; in 22 of the remainder they disappeared within 2 days, and in the 3 others in 3-6 days after the injection. None developed cholera. The numbers are small, but in another hospital several carriers continued to excrete the organisms for 15 days, and at least 2 developed cholera and died; and the authors consider that the treatment is worth a further trial. In none of their cases could they detect the presence of cholera antibodies in the serum of the patients treated.

J. H. S.

**EMMERICH (Rudolf) & JUSBASCHIAN (A.).** *Die Beeinträchtigung des Gift- i.e., Nitritbildungsvermögens der Choleravibrien durch freie salpetrige Säure.* [The Influence of Free Nitrous Acid on the Power of the Cholera-Vibrio to produce Poison, *i.e.*, Nitrites.]—*Arch. f. Hyg.* 1912. Vol. 76. pp. 12-76.

In accordance with the teaching of KOCH it is generally held at the present time that the factors essential for an epidemic of cholera are present if we are given a case of the disease and some means of transmitting the organisms from that case to other persons. The transmission may be direct through contact with the excreted vibrios or indirect through contamination of water, milk, food, &c.; but whatever the method of transmission the organisms once transferred are as capable of producing the disease



in the second individual as they were in the first. This is denied by **EMMERICH** who has developed and modified the views of **MAX PETTENKOFER**, and in the present paper he restates his position with some fresh experimental evidence on the production of nitrites by cholera-vibrios. Unlike **PETTENKOFER**, **Emmerich** admits that the transference of the excreted unmodified vibrios from man to man can give rise to choleraic disturbance. The disease so produced, however, is not virulent cholera: it is a cholera of mild type, practically never fatal, and the true cholera epidemic with its 40-50 per cent. mortality is never due to man to man infection of this character. He bases this opinion on the numerous instances he has got together of cholera outbreaks where the infection must be due to excreted organisms, *e.g.*, ship-epidemics, infection of attendants on the sick, accidental or intentional infection by drinking cultures or stools (he has collected 53 such instances), in all of which the disease takes a very mild type with very low case-mortality, and he includes under this head town-epidemics occurring in wet seasons. Virulent cholera, he maintains, is caused only by organisms which have come from the soil, and only from soil which has been dry for lengthy periods, during which a gradual accumulation of suitable food has been collected on the surface by capillary upflow from the deeper layers of the ground. Excreted organisms, he asserts, have an enfeebled vitality and a greatly diminished power of nitrite-formation. When a case of the disease is imported into a district it can give rise only to mild forms unless the excreted organisms reach a suitable soil. When this happens the organisms regain their full powers, and from such foci the true epidemics are derived by repeated infection with these soil-bacteria, which are distributed by wind, the feet of passers-by, &c.

The severity of an infection with cholera depends on the capacity of the vibrio to produce nitrites and nitrous acid from the nitrates or other food, the cholera poison being simply nitrite or nitrous acid. These poisons occur regularly in fatal doses in the faeces and urine of cholera cases, and even after death the contents of the small intestine are acid in half the cases and contain nitrous acid; the anatomical changes and the clinical symptoms are due solely to their action, and can be reproduced by them in rabbits or guineapigs; cholera always begins after taking food in large quantities, and the only group of human beings immune to the disease are sucklings which take no nitrate in their food. The experiments recorded in this paper are directed to establishing the facts that excreted organisms have lost much of their power of producing nitrites, but regain it on passage through suitable soil; and he shows that exposure of these fully active organisms to nitrites in conditions designed to simulate those occurring in the body takes away again much of their power of producing nitrites.

J. H. S.

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## MISCELLANEOUS.

BISHOPP (F. C.) & KING (W. V.). **Additional Notes on the Biology of the Rocky Mountain Spotted-Fever Tick.**—*Jl. Econ. Entomol.* 1913. April. Vol. 6. No. 2. pp. 200-211.

The splendid work of the late H. T. RICKETTS during 1906 and the three succeeding years, which showed that the tick *Dermacentor venustus* is the carrier of Rocky Mountain spotted fever, is first referred to. The fight against the disease, as that authority indicated, resolves itself into a campaign against the transmitting agent. The authors therefore go into the life cycle of the tick in detail.

As the adult tick is, with rare exceptions, the only stage which attacks man, the most feasible method of prophylaxis is to destroy it on the domestic animals. The determination of the period of activity of this stage is therefore of importance. The adults of *Dermacentor venustus* first appear after the first warm days of early spring (March eleventh and fifteenth in 1910 and 1911 in the Bitter Root Valley for example). Their date of disappearance in the autumn is much more variable, but after the first of August they do not appear anxious to feed and endeavour to escape from the host when applied. The months of their greatest abundance are April and May.

The mountain goat serves as a host for a considerable number of the adults, while the immature stages may also develop on this animal or on small mammals occurring on the mountains inhabited by the goats. It is possible that the goat is a natural reservoir for the disease virus.

All stages of the tick—larvae, nymphs and adults—hibernate through the winter. Where this takes place has not been accurately determined, though it has been suggested that the different stages are probably carried into the winter quarters of ground squirrels and other animals.

The question of the time required for the *Dermacentor venustus* to complete its life cycle has been considerably discussed. At first it was thought that one generation developed annually, but more recent investigations have shown that this is not the normal cycle, two years being the usual time required. The adult, under natural conditions, does not become engorged the same season as it became transformed into that stage; therefore the completion of a cycle developing from overwintered nymphs is unlikely. Similarly the same habit prevents the completion of a generation from overwintered larvae. The principal hosts for the developing stages are the ground squirrel, *Citellus columbianus*, and the woodchuck, *Marmota flaviventris*, these rodents going into hibernation about the middle of August. In an experiment sixty-seven nymphs were placed on hosts on August 17 and 18 1910. These did not transform to adults till the following summer; hence they required more than one year for completing the cycle.

The authors believe then that the possibility of the completion of a cycle in one season is very remote in the Bitter Root Valley,

though in more southern localities there is more likelihood of some ticks, which hibernate as adults, being able to do so. At the rate of development noted by them in the Bitter Root Valley it is possible that the cycle may even take three years for completion.

In addition to spotted fever a considerable number of curious cases of paralysis, more or less complete and in some cases fatal, have been reported as being produced by *Dermacentor venustus*. [See papers below by TODD and EATON.]

Nine cases, in each of which a tick (in one case two ticks) was found attached to the hair below the occipital prominence, have been reported by TEMPLE from Oregon, while a similar case has been reported from Wyoming and one or two have been heard of in Montana. The affection, as reported, first manifests itself as a partial paralysis of the lower limbs, the paralysis advancing to the upper part of the body and arms, ultimately affecting speech, deglutition and respiration. The symptoms subside rapidly after removal of the tick. The point of attack seems to be an important factor in producing the symptoms. These observations, of course, by no means show conclusively that the condition has anything to do with the presence of the tick. Local, itching sores frequently follow at the point of attachment, especially if infection is introduced by scratching. Swelling of a limb and lameness have been known to persist for weeks following the bite of a spotted-fever tick which was left attached for some time.

G. C. Low.

TODD (John L.). i. **Does a Human Tick-Borne Disease Exist in British Columbia?**—*Canadian Med. Assoc. Jl.* 1912. August. Vol. 2. (new ser.) No. 8. p. 686.

ii. **Tick Bite in British Columbia.**—*Ibid.* December. No. 12. pp. 1118-9.

i. The author refers to Rocky Mountain spotted fever and states that the tick which transmits this disease in Montana exists also in some parts of British Columbia. In order to ascertain whether the disease occurs there Todd sent letters to a few doctors asking them whether they had ever met with cases of illness which had been ascribed to tick bites. Among the replies were letters from two doctors, who described several cases in which symptoms entirely different from those seen in Rocky Mountain spotted fever of Montana were ascribed to the bites of ticks. The history in all was practically identical, viz., paresis or paralysis which came on suddenly. On examination a tick was found attached to some part of the patient's body, in most instances near the nape of the neck. Some of the cases died; while in others after the tick was removed complete recovery followed in a few days.

The similarity of the history in the cases reported was striking and replies to a letter sent by Todd to different doctors revealed the fact that cases with similar histories have been seen in seven places in the southern and central parts of British Columbia.

On this evidence the author believes that it is possible that an undescribed disease may be caused by ticks in British Columbia.

ii. As a result of the letters sent out by the author forty replies were received. Some of the writers described cases in which infection of the wound caused by the tick's bite had been followed by a severe local inflammation, while others mentioned instances in which the bites of ticks had been followed by paresis or paralysis and sometimes by death. A description of these is given.

One case recorded by NELSON was that of a child who died suddenly with symptoms of acute ascending paralysis. After death a large tick was found at the nape of the neck. In 1901 a child, with the same symptoms, died after an illness of two days. A tick was found attached to the right temple. The knowledge of these two cases suggested the presence of a tick when a third child, previously very healthy, was seen, whose legs had been becoming weaker for two days. One was found at the nape of the neck; it was removed and in two days the child was quite well again. In April 1912, a girl of three had become paralysed. Paralysis of the legs was complete and the reflexes were gone; paresis of the arms was marked. Three ticks were removed from the nape of the neck and the child recovered completely.

A consideration of these different reports indicates that severe symptoms may follow the bites of ticks in British Columbia; children seem to be most affected. In them paresis and paralysis of the extremities, especially of the legs, are the most constant symptoms. Up till the time of Todd's investigation into the subject, it was not known that the bites of ticks in British Columbia could produce such symptoms.

G. C. L.

EATON (E. M.). **A Case of Tick-Bite followed by Wide-spread Transitory Muscular Paralysis.**—*Australasian Med. Gaz.* 1913. April 26. Vol. 33. No. 17. (No. 432.) pp. 391-394.

The case was that of a girl of four and a half years of age who had been in good health until the day prior to the author seeing her. On that day she would take no food and in the afternoon became restless and unsteady on her feet. When she was undressed and put to bed a tick was found with its head embedded on the back of the right shoulder. The parents cut the body off, leaving the mouth parts *in situ*. From the description given by the parents Eaton believes that the parasite was either *Ixodes ricinus* or *Ixodes holocyclus*.

On the second day the child vomited three or four times, and as she could not stand and appeared very ill, she was sent to the author, who found her in a state bordering on delirium, but sufficiently conscious to be annoyed by examination. The temperature was 101.4° F., the pulse rate 132, and the respirations 36 per minute. The muscles of the legs and thighs were quite flaccid and the lower limbs motionless, save for movement which could still be made out at the hip joints: the arm muscles could be moved, but with little force. The knee-jerks were absent.

Over the lower part of the right scapula was a pink patch about the size of a penny, in the centre of which was a purplish black spot, a quarter of an inch in diameter. In the middle of this was an aperture in which lay the head parts of the tick; these were scraped out and the cavity cleansed with pure carbolic.

On the third day there was diarrhoea but no abdominal pain and no vomiting. There was no sign of return of voluntary movement to the legs but the muscles were not so limp. On the next day, the fourth, the knee-jerks were obtainable but with difficulty; the child now could stand and even walk a few steps with support. The pupils were still inactive to light. Diarrhoea continued but was less severe.

On the fifth day the child could walk without support. The discolouration of the bite mark had become reduced and the cavity was healing up. This was the last time the author saw the patient but he was informed that progress to complete recovery within the next few days was uninterrupted.

He quotes other cases noted by CLELAND\*; in one of these amblyopia was the most striking symptom, the pupillary movements being normal while only slight muscular weakness was present. In another (an infant of thirteen months), which proved fatal within two days, paralysis was present in the legs and respiratory muscles. In a third case faintness commenced within an hour of the attachment of the ticks. There was no paralysis but severe cardiac symptoms lasted over a week.

CLELAND also refers to a statement of BANCROFT's that the bites of Queensland ticks frequently kill dogs and cats. The symptoms in dogs are epileptiform attacks or prolonged convulsions while peripheral paralysis has also been seen during convalescence. One attack is said to confer immunity.

Eaton draws attention to the fact that the symptoms bear a very close resemblance to those of conine poisoning. As to the source of poison, he believes there are three possibilities:—(1) its pre-formation by the tick, (2) that it might be an infection conveyed by the tick, (3) that the poison might be elaborated locally after the bite.

[From the above description these cases of tick bite disease would seem to be not infrequent in Australia, and as TODD's cases from British Columbia, and BISHOPP and KING's from Montana would seem to be identical, the infection is evidently wide-spread, occurring in different parts of the world.]

G. C. L.

GRAY (Douglas G.). *Climatic Bubo*.—*China Med. Jl.* 1913. May. Vol. 27. No. 3. pp. 180-184.

The author states that the superficial oblique inguinal glands are the ones affected in climatic bubo. The glands of this group are disposed irregularly along Poupart's ligament and receive the lymphatic vessels from the integument of the scrotum, penis,

\* *Australasian Medical Gazette*, 1912. Sept. 21. Vol. 32, No. 12 (No. 401), pp. 295-296.

parietes of the abdomen, perineal and gluteal regions, and the mucous membrane of the urethra. All the cases, 27 in number, were seen in men in the prime of life, between the ages of 22 and 57 years, mainly the class who are prone to promiscuous intercourse. The author never met with it in the female sex, this observation agreeing with the reports of others who have studied the subject. The glands on removal were found to be massed together and enlarged, many of them showing more or less advanced areas of necrosis.

As regards treatment the author believes that medicines are of little or no use. Iodides, quinine, arseno-ferratoze, etc., were all tried but with no noticeable effects. Similarly "606" intravenously, and a course of mixed staphylococcic vaccine were of no avail. He therefore advises early operation. Every gland that can be palpated must be removed and if done early enough this is easy as the glandular tissue is only slightly inflamed and the subcutaneous tissue not adherent. After extirpation, deep sutures reaching to the floor of the cavity are put in to approximate the walls. A drain may be left in for two or three days.

G. C. L.

TRIADO (A. J. J.). **Tropical or Climatic Buboes.**—*Australasian Med. Gaz.* 1913. May 10. Vol. 33. No. 19. (No. 434.) pp. 442-443.

In the last nine years the author has seen fifty cases of climatic bubo. He believes that sexual connection between aboriginal women and white men has to do with their production. [See also this *Bulletin*, Vol. 1, p. 474.] All the cases seen were in white males; natives both male and female seem to be exempt (two hundred and fifty cases examined). In twenty-seven the left inguinal glands were affected and in the remaining twenty-three the right. Thirty-seven had had gonorrhoea on some previous occasion of varying duration. Two had had gonorrhoea and syphilis, both in the past. The size of the buboes varied up to that of a hen's egg. After removal the different glands were seen to be distinct, all of them, especially the more superficial ones, showing softening in their centres. Periadentitis occurred, varying with the chronicity of the complaint. Pain was not a specially marked feature, but when the glands were very large they caused a sense of tightness in the groin and consequent difficulty in walking.

The diagnosis of these buboes from syphilitic and gonorrhoeal ones, Triado believes, offers no difficulties, their history and clinical course being quite different. Gonorrhoeal buboes show early tendency to suppuration and spontaneous bursting through the overlying skin if not early incised. In syphilis the bilateral distribution and the characteristic hardness of the bubo and other signs make the differential diagnosis easy. In all the author's cases the buboes were unilateral, and up to the time of writing none had developed a bubo on the opposite side.

The treatment adopted was complete removal in one mass of the whole of the affected glands, the earlier the better, as the

cavity left in the groin then is smaller. Occasionally complete removal in one mass is difficult, so in these cases curettage has to be adopted. After the removal the cavity is swabbed out with pure carbolic acid. There were no recurrences, the results of the operation being perfect.

Fifteen of the cases were examined bacteriologically, but no definite micro-organism was found. [Absence of bacteria or of any other parasites is what has been noted by various authors previously.]

G. C. L.

SAMBON (Louis W.). *Porocephaliasis in Man.*—*Jl. Trop. Med. & Hyg.* 1913. Apr. 1. Vol. 16. No. 7. pp. 97-100.

The first part of the paper completes the description of *Porocephalus clavatus* and is a continuation of a paper which appeared in the *Journal of Tropical Medicine and Hygiene*, December 16, 1912. (See this *Bulletin*, Vol. 1, pp. 403-405). Two other porocephali, *Porocephalus stilesi* and *Porocephalus wardi*, are now described. The former species was collected by NATTERER in 1821 at Ypanema (South America) from the lungs and body cavity of *Bothrops jararaca* (= *Lachesis lanceolatus*). The adult forms are found in the lungs of *Lachesis mutus* (Linn.), the Surucucu or Bush master, and of *Lachesis lanceolatus* (Lacep.), the rat-tailed pit-viper or Fer-de-lance. The nymphal form is unknown. The geographical distribution is Central and Tropical America.

Though more uniform in diameter throughout and with a more rounded terminal swelling, *Porocephalus stilesi* is similar in form of body and general appearance to *Porocephalus crotali* (*sensu stricto*) and *Porocephalus clavatus* Wyman, but differs from both in the number of annulations. These, which are over 80 in *Porocephalus crotali* and only about 40 in *Porocephalus clavatus*, number from 45 to 50 in *Porocephalus stilesi*.

*Porocephalus wardi* (Sambon, 1909) was found by NATTERER in the abdominal cavity of a *Podinema teguexin* (*Tupinambis teguexin*). Two males and one female were found 3 in. to 6 in. long. They are mentioned by DIESING and ascribed to the species *Porocephalus crotali* found by HUMBOLDT in *Crotalus terrificus*. Sambon, however, considers that this is a new species and has given it the above name. Its geographical distribution is South America.

G. C. L.

FRANÇA (C.). *Un Cas de Chappa?*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 351-355.

The case resembled very closely one of gout, but the author believes that he could identify it with an infection described by ~~Read~~ under the name of Chappa.\* The patient acquired the infection in Mozambique.

\* *Jl. Trop. Med. & Hyg.* 1901. Oct. 15. Vol. 4. pp. 333-334.

Chappa, according to READ, is characterised by the following symptoms:—The illness commences by severe pains in the limbs with swelling of the articulations which give the sense of fluctuation. A little afterwards numerous nodules appear on different parts of the body; the skin which covers these nodules ulcerates without the formation of abscesses. In one case READ opened a joint on account of the fluctuation, but only found a fatty looking substance. The disease ends by attacking the bones and disorganising the articulations.

The differential diagnosis from gout is relatively easy, according to França, firstly by the analysis of the matter which infiltrates the tissues (protein) and secondly by radiography. Contrary to the tophus of gout (urates) the accumulations of protein are little permeable to the Röntgen rays and in the radiograph give a somewhat dark spot whilst urates give a clear spot.

G. C. L.

PHISALIX (Marie). *Propriétés Vaccinantes du Venin Muqueux de la Peau des Batraciens contre lui-même et contre le Venin de la Vipère Aspic.*—*Bull. Soc. Path. Exot.* 1913. Mar. Vol. 6. No. 3. pp. 190-195.

The analogy which exists between the physiological action of the secretion from the cutaneous mucous glands of batrachians and that of the serum of the eel and hedgehog, and of the venom of bees, spiders, and vipers has prompted the author to try some vaccination experiments with the former both against itself and against the venom of the viper. Frogs, guinea-pigs, and rabbits were used, the mucous venom of the terrestrial salamander (*Salamandra maculosa* Gray), the green frog (*Rana esculenta* Günth), and other batrachians being employed for the inoculation.

In the rabbit the most rapid method of immunisation is by direct inoculation into the veins, but as the action of the venom in certain cases is fulminating (foudroyante) (venom of *Alytes obstetricans*, *Discoglossus pictus* and *Rana esculenta*) it is necessary to attenuate the virus by appropriate heating. Finally the rabbit develops a certain degree of immunity against viperine poison. The practical value of the method is that it may be applied to young dogs, which are often killed by viper bites in their first season of hunting.

G. C. L.

BAUJEAN (R.). *Note sur le Venin de Bitis arietans ou Vipère heurtante.*—*Bull. Soc. Path. Exot.* 1913. Jan. Vol. 6. No. 1. pp. 50-54.

Observations were made on a small quantity of dried puff adder venom sent by FITZSIMONS from Port Elizabeth, Natal to CALMETTE in Lille. The toxic, proteolytic, haemolytic and coagulating properties of the venom were tested. The chief characteristics of the poison were its intense proteolytic action and the



haemorrhagic lesions which it provoked in animals. It also showed the following peculiarities—(1) An absence of all haemolytic action. (2) A strong haemorrhagic tendency not destroyed by a temperature of a 100° C. maintained for half an hour. (3) An absence of coagulation of the blood on intravenous injection, and of normal plasma and blood from the head of the leech in vitro, although on the contrary it coagulated citrated and oxalated blood.

G. C. L.

WILLETS (David G.). General Conditions affecting the Public Health and Diseases prevalent in the Batanes Islands, P.I.—*Philippine Jl. Sci.* Sec. B. Trop. Med. 1913. Feb. Vol. 8. No. 1. pp. 49-57.

The author's report is based upon observations made in the Batanes Islands, a group lying between Formosa and the Philippines, chiefly in the town of Santo Domingo de Basco, Batan Island, from April 2nd to May 7th, 1912.

A disease resembling malaria in some respects was met with. It was characterised not only by fever but also by chills, vomiting, and sweating. However, in the examination of a number of persons from Itbayat, only one enlarged spleen was met with, and the blood of this patient, as well as that of a number of others from the same place and a few from Batan, who claimed to have had the fever within the past two years, was examined for malarial parasites with negative results. Further investigation is evidently necessary to prove what this fever is.

No case was seen suggesting elephantiasis, and a search for filariae in the blood of 191 adults taken at night resulted negatively.

Acute cases of dysentery were not met with and only two chronic ones, neither of these showing entamoebae. In stools five infections of protozoan parasites were found, one with monads, two with entamoebae and two with *Balantidium coli*.

Evidence of helminthic infection was readily found. 400 persons were examined with the following results. Single infections were present in 46 per cent., double in 42·5 per cent., triple in 11·5 per cent., *Ascaris* in 92·8 per cent., *Trichiuris* in 46·7 per cent., hookworms in 24·5 per cent., *Oxyuris* in one per cent., and *Strongyloides* in 0·5 per cent. No cestode infections occurred in the 400 examined, but one infection with *Taenia saginata* was found at Sabtang. Of other tropical diseases cholera was present in 1902, dengue fever occurs from time to time and beriberi is apparently absent. No cases of yaws were seen.

Since 1906 several lepers have been taken from the Islands; some of these were natives, others fugitives from northern Luzon.

Venereal diseases are apparently rare.

G. C. L.

**Photomicrographs of Spirochaetae, Entamebae, Plasmodia, Trypanosomes, Leishmania, Negri Bodies, and Parasitic Helminths.**—*U.S. War Dept. Office of the Surgeon General. Bulletin No. 1. 1913. Jan. 46 pp. With 17 plates. (Washington: Govt. Printing Office.)*

This Bulletin contains a very interesting series of microphotographs of parasites from different tropical diseases. The negatives have been made mainly from specimens in the Army Medical Museum and though the collection is in no sense a complete one, yet it is believed that the photographs will be of considerable help to medical officers in the study and diagnosis of the different parasites. The photographs are clear and perfectly executed. An explanatory text accompanies them, the methods used in the preparation of these specimens being given and ample directions for the preparation of similar specimens.

The greater number of the negatives are the work of the late Dr. W. M. GRAY of the Army Medical Museum, while the text is the work of Capt. C. F. CRAIG, Capt. H. J. NICHOLS, and Major F. F. RUSSELL.

G. C. L.

**HUMMEL (E. M.). The Prevalence of Asthenic Disorders of the Nervous System in Warm Climates.**—*Interstate Med. Jl. 1913. June. Vol. 20. No. 6. pp. 522-525.*

The author states that during his practice of neurology in a sub-tropical climate for a number of years he has been impressed with the great frequency of those types of neuroses which are regarded as founded upon asthenic states of the nervous system, and which we usually designate as neurasthenic. The predominance of this type of neurosis is out of keeping with what is observed in the larger nerve clinics of the United States and of Europe, which are all situated in colder climates. So prevalent are such neuroses in the Southern section of the States that the author believes climatic conditions must be responsible for their development. The condition varies from a simple lack of energy to a pronounced neurasthenia and in most instances no definite physical cause can be blamed for it. People of blond complexion suffer most frequently.

WOODRUFF of the United States Army, who has made extensive observations on the subject, is specially convinced of this. Contrary to accustomed belief it is not so much the high temperature and humid condition of the atmosphere which depresses and enervates those exposed to tropical climates as the intense light of the sun that acts in a deleterious way. According to the author the best proof of this is the fact that all aboriginal people in tropical or sub-tropical climates are highly pigmented and he states that there are indications that all the aboriginal races of the earth were at least brunette in type.

Either directly or indirectly as a result of the excessive action of light rays on the nervous system, the digestive functions become disordered or reduced in activity. This has led to the common practice of the excessive use of peppery condiments and alcoholic appetizers, equally harmful practices. The excessive consumption of food should also be avoided. The best guide to the quantity required is the appetite and not irritating condiments and alcoholic drinks. Alcohol disturbs not only the liver and stomach but the nervous system and it is a well known fact that alcoholics are especially prone to develop sunstroke.

G. C. L.

**LE ROY DES BARRES.** *Rapport sur la Natalité, la Morbidité et la Mortalité à Hanoi en 1912.*—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. Vol. 4. No. 5. pp. 248-251.

This interesting report states some of the chief causes of death met with in Hanoi in 1912. During the year out of 1,833 deaths 71 were in Europeans. Malaria was responsible for 185 deaths, ten of these being in Europeans. Relapsing fever gave 46 deaths out of 437 cases. This high percentage of death, it is pointed out, is due to the fact that "606" was not used in the treatment or only in the last stages. Tuberculosis was responsible for a large death rate, 152 deaths; 136 of the cases were of the pulmonary type. The author states that this disease is very frequent amongst the Annamites, and that it will be necessary to take measures to prevent its diffusion.

One case of plague and none of cholera were observed during the year.

G. C. L.

**WURTZ (M.).** *Moustiques et Fosses d'Aisances.*—*Rev. de Méd. et d'Hyg. Tropicales.* 1913. Vol. 10. No. 1. pp. 13-15.

The author was called upon to investigate an invasion of mosquitoes in a hospital (*l'Hospice de Vieillards des Ménages*) of which he was the physician. It was found that the insects were breeding in two cesspits situated in the grounds of the building. A chemical examination of the sewage gave the following:—

Reaction to litmus	...	...	alkaline.
Phenol-phthalein reaction	...	...	alkaline
Amount of alkalinity	...	...	0.44 gm. per litre (expressed in NaOH)
Density	...	...	1001
Chlorides, about	...	...	0.55 gm. per litre
Nitrogenous substances, total nitrogen	...	...	0.39 gm. per litre
Albuminoid substances	...	...	traces
Ammonia	...	...	0.37 gm. per litre
Carbonates	...	...	traces
Organic substances.—Reducing action on Fehling's solution	...	...	almost none
Reducing action on acid permanganate	...	...	perceptible

The application of petroleum to the cesspits was immediately followed by a cessation of the insects and by systematical oiling they were easily kept under.

G. C. L.

**FERREYROLLES (Paul).** **La Destruction Pratique des Moustiques sous les Tropiques.** (L'Organisation sanitaire de Panama. Les Résultats acquis.)—*Rev. de Méd. et d'Hyg. Tropicales.* 1913. Vol. 10. No. 1. pp. 16-23.

An account is given of sanitary organisation in Panama and the results obtained from the measures adopted to destroy mosquitoes. Attention is drawn to the value of small fish as destroyers of larvae, but it is pointed out that these are only useful where larvicide cannot be employed.

[The methods adopted in Panama are now so well known that they need not be given in detail here. The part played by small fish in the destruction of mosquitoes has probably been much magnified.]

G. C. L.

**KNAB (Frederick).** **Spider's Web and Malaria.**—*Jl. Trop. Med. & Hyg.* 1913. May 1. Vol. 16. No. 9. pp. 133-134.

The author, replying to a paper by O'CONNELL who pleads for the web-making spiders as effective destroyers of malaria-transmitting mosquitoes, points out that records of the supposed efficiency of spiders as mosquito destroyers are not sometimes based upon actual observations. SAMBON and LOW have shown that the *Anopheles maculipennis* in Italy is often found resting on cobwebs in houses, stables and hen-houses in great numbers, and now Knab demonstrates that *A. quadrimaculatus*, the North American representative of the European *A. maculipennis*, also does the same. From these observations it is clear that web-making spiders in Italy and North America do not act as effective destroyers of malaria bearing mosquitoes. It is of course, as the authors says, possible that in other parts of the world other species of *Anopheles* may be caught in spiders' webs but, if so, this has not been demonstrated. The author's conclusions are that "the generalizations, unsupported by sufficient and accurate data, with which economic mosquito literature now abounds, are likely to prove valueless and indeed misleading."

G. C. L.

**JACKSON (Thos.).** **Cocoonut Oil as an Insecticide.**—*Indian Med. Gaz.* 1913. May. Vol. 48. No. 5. p. 203.

Jackson recommends cocoonut oil as an insecticide for bugs. He states that all that is necessary is to smear a little of the oil over the place where these insects are found, or where there are cracks and crevices the oil may be dropped into them. By the

use of this medium any barrack or hospital ward or article of furniture or bedding can, in a short time, be freed of bugs and with a minimum amount of disturbance or upheaval. The author refers to RUCKER's work on the destruction of bugs by fumigation (this *Bulletin*, Vol. I., pp. 533-534) but queries whether the ova could be destroyed by the different gases used. Cocoanut oil will, however, Jackson says, destroy the ova as well as the adult insect.

G. C. L.

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## TROPICAL DISEASES BUREAU.

TROPICAL DISEASES  
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[No. 5.]

## MALARIA.

## EPIDEMIOLOGICAL AND CLINICAL.

LEGER (Marcel). *Le Paludisme en Corse. Recherches Microbiologiques. Etudes Prophylactiques.*—*Publication de l'Institut Pasteur.* 1913. 60 pp. With 2 maps and 7 tables. (Laval: L. Barneoud et Cie., Imprimeurs.)

Malaria in all its forms is the principal disease in Corsica. It is responsible for the loss of over 1,000,000 days of work yearly. In the village of Pietra di Verde four-fifths of the inhabitants were found to be infected. The case incidence varies in different parts of the island and the disease varies in intensity in the different months of the year. The East Coast is unhealthy over all its extent from Bastia to Bonifacio, and there are scarcely any privileged places in the whole island where the disease is exceptional. It is common along the courses of the rivers. Ponte-Lecia on the river Golo, 40 kilometres inland and 200 metres above sea level, is very malarious; even Corti in the very heart of the island situated at a height of 500 metres is not exempt. Important malarial foci also exist at the mouths of certain streams on the West Coast at Saint-Florent, Galvi, Ajaccio, Porto-Pollo and Pianottoli. The primary attacks of fever occur in June and increase progressively in number in July, August, September and October. The disease diminishes in November and is exceptional from December to May.

The author gives a detailed account of his investigations regarding the breeding places of Anophelines in the island, and the parasitic and splenic indices of the inhabitants. In the months of May and June, out of a total of 1,031 people examined, the parasitic index in the blood was 7.56 per cent., the majority of the cases being benign tertian. The spleen rate was 28.8 per cent. During the hot months of July, August and September, the blood index was 16.49 per cent. and the spleen rate 42.17 per cent. Benign and malignant tertian cases, during these months, were equally common and quartan also occurred. After the hot season

in November, the blood index fell to 6·5 per cent. and the spleen rate to 31·95 per cent.; during this period malignant tertian was most prevalent.

In the second part of his report the author describes the anti-malarial measures carried out. He states that the usual method adopted in Corsica in the struggle against malaria is a general exodus of the peasants and tradespeople from the plains into the mountains during the hot months. During their stay in the mountains they are badly housed and very badly fed, and spend the entire savings of their year's work. This migration from the plains commences in June and they return again towards the end of September, or later if their resources can hold out. In spite of this they do not escape malarial infection. A Corsican anti-malarial league has for some time been established to educate the people in the part played by mosquitoes in the disease. Prophylactic quinine has been given and a few houses have been mosquito-proofed. These endeavours have been attended with some success; but an enormous amount still remains to be done. The author advises as to what further measures should be adopted.

In the third part of the report the results obtained by systematic quinine prophylaxis are described. During the summer of 1912 a number of quinisation areas were established in order to prove to the peasants that it was possible to withstand the dangerous season without migrating into the mountains, by taking regularly small doses of quinine salts. He describes the results obtained in five different areas. In the area of Casabianda the blood index in May was 42·85 and the spleen rate 57·14 per cent. Among those who were given quinine the blood index fell to 9·09 and 7·14 per cent. in August and November respectively and the spleen rate fell to 9·09 and 7·14 per cent. The quinisation was commenced on the 10th of June. Each person received daily 0·2 grammes of quinine bichloride. To children under 5 years were given chocolate tabloids containing 0·15 grammes of the drug. This medication was continued till the 15th day of October.

[The report is a very excellent one and those wishing for further information should consult it.]

D. Thomson.

SERGEANT (Edmond & Etienne). *Etudes Epidémiologiques et Prophylactiques du Paludisme. Neuvième et Dixième Campagnes en Algérie, en 1910 et 1911.*—*Ann. Inst. Pasteur.* 1913. May 25. Vol. 27. No. 5. pp. 373-390.

The authors state that malaria was much less severe in 1910 than in 1909 in Algeria, but more severe again in 1911. In a number of places malarial epidemics occurred, especially in Mitidja. In the valley of the Seybouse the malarial season lasted until April 1912. In 1911 mosquito breeding pools were more abundant and persisted longer than in the preceding years, as a result of the rains in the spring time continuing longer than usual. The summer of that year was extremely hot and prolonged. Extensive land excavations during

the construction of defence works may also have accounted for the increase of malaria in 1911.

In 1910 the spleen rate was 16·9 per cent. in a total of 3,847 persons examined, and parasites were found in the blood of 8 cases out of a total of 52 examined. In 1911, the spleen rate was 23·5 per cent. (3,570 persons examined) and 19 out of 50 cases presented parasites in their blood. In spite of the increase of malaria in 1911, it was not found that blackwater fever was more prevalent during that year.

Regarding the antimalarial work in progress, they state that the antilarval measures, commenced in 1909 at Montebello and other localities, have given excellent results. These measures consisted in filling in and drainage. Adult mosquitoes are destroyed in dwellings by the combustion of simple pyrethrum powder. Mosquito traps are used and good results have also been obtained by alcohol vapour. Quinine has been given prophylactically by three different methods: (a) daily doses of 20 centigrams; (b) doses of 60 centigrams every three days; (c) doses of 60 centigrams every six days. Children receive smaller doses in proportion to age. All these methods have given appreciable results in reducing the spleen rate. The first apparently gave better results than (b) and (c). The bihydrochlorate of quinine is employed, in tabloid form. Three thousand people are given quinine regularly every year by the quinine dispensers. The quinisation has been confined to certain demonstration areas and to localities where malaria is especially severe. In 1910 and 1911, sixteen quinine dispensers (European) were employed—eleven men and five women. Six teachers have administered quinine to the children in their schools in the springtime and autumn.

Every year the number of mosquito proof dwellings is increasing and a considerable amount of literature regarding the prevention of malaria is distributed amongst the inhabitants. In the demonstration areas good results have been obtained. In 1910 at Montebello the number of protected Europeans was 78. Among these there were no cases of primary infection and very few relapses in those who had already suffered from malaria. Among those who were not protected malaria increased and there were at least 16 cases of primary infection. In 1911 similar results were noted.

At Mondovi in 1910 there was no case of primary infection in 800 protected Europeans and only one case of mild blackwater fever in a patient who had previously suffered from malaria. Out of 362 unprotected Europeans there were two severe cases of blackwater fever. 1911 (Mondovi), 900 protected Europeans—2 primary infections and 3 severe cases of blackwater fever in 3 children who did not receive quinine. Among 400 unprotected Europeans—six severe cases of primary infection.

Tourville, 1910, 1,000 protected Europeans,—no case of primary infection. Unprotected,—2 cases of primary infection



out of 4 persons. 1911, 1,000 protected Europeans,—no cases of primary infection. Unprotected,—8 cases out of 100 persons.

Sainte Leonie, 1910, 300 protected Europeans,—no cases of primary infection. Unprotected,—2 cases in 4 persons. 1911, 300 protected Europeans—no cases of primary infection. Unprotected—2 cases in 4 persons.

In 1910 and 1911 850 kilogrammes of quinine tabloids were distributed for prophylactic and curative purposes.

D. T.

O'CONNELL (Matthew D.). **The Meteorology of Malaria.**—*Jl. Trop. Med. & Hyg.* 1913. June 2. Vol. 16. No. 11. pp. 165-166.

The author maintains that the atmospheric conditions at Puttalam and at Kurunegala in Ceylon during October are somewhat similar as regards humidity and temperature to the conditions in the Lancashire cotton sheds. Since it has been shown that the body temperature of workers in the latter are raised above normal, he thinks that the atmospheric conditions in the former must necessarily raise the temperature of the inhabitants to about 100° F. He considers that such a state of affairs is conducive to an outbreak of malaria.

D. T.

JAMES (W. M.). **Notes on the Etiology of Relapse in Malarial Infections.**—*Jl. of Infectious Diseases.* 1913. May. Vol. 12. No. 3. pp. 277-325. With 1 coloured plate.

The author asserts that a considerable part of the malaria in the Panama Canal Zone is due to relapses. It has been shown there that the increase in the amount of malaria at the beginning of the wet season is out of proportion to the increase in the number of *Anopheles* mosquitoes. This shows a relationship between exposure to wet and chilling and the increase in the malaria rate. The wet and chilling no doubt bring on relapses. Relapses are more liable to occur in aestivo-autumnal than in benign tertian infections; but they are most frequent in quartan malaria. Relapses have followed every method of giving quinine, though in varying proportions, and until the true cause of relapse is known the treatment of malaria will remain to some extent empirical and imperfect.

The hypotheses as to the cause of relapse fall under two heads:—(1) That it is due to the development of a resistant and latent form of parasite different from the asexual type. This peculiar form in conditions not clearly understood transforms itself into the asexual type and begins a relapse. This hypothesis includes the theory of parthenogenesis in the female gametes as advanced by CANNALIS, GRASSI, MAURER, NEEB, SCHAUDINN, and HARRISON. CRAIG holds that relapses are due to a resistant form of parasite formed by the conjugation of two young asexual forms and states that this resistant type

can under certain conditions split up into asexual parasites. EWING also believes in the existence of conjugation forms. (2) That relapses are due to the ordinary asexual parasites. After treatment, or spontaneously, these parasites may disappear, but the disappearance is only apparent; a few of them, not numerous enough to be detected, remain and continue their cycle. Under certain conditions these few are able to increase in number until the clinical symptoms of relapse are manifested. This hypothesis has the support of ROSS, THOMSON, and BIGNAMI.

With regard to the first hypothesis very few observers have ever seen parthenogenesis or the so-called resistant forms, but the fact that gametes are very resistant to quinine favours the belief that these by reverting into the asexual form may cause relapses. In spite of the search for parthenogenesis by many competent observers there is still very limited evidence that such a process occurs. BIGNAMI, FÜLLEBORN and the author believe that the so-called parthenogenetic forms seen and described by SCHAUDINN and others are merely atypical sporulating asexual parasites such as are seen in anaemic blood and during quinine treatment. At any rate after a careful examination of the data it cannot be held that parthenogenesis is an established fact, and there is still less evidence to show that this supposed phenomenon is the cause of relapses. THOMSON has shown that individual gametes do not persist for long periods in the blood. The continual persistence of gametes means that the asexual cycle which produces the gametes is still going on. So that when gametes persist day after day it is certain that an asexual cycle is simultaneously pursuing its development, perhaps in the internal circulation.

With regard to the second hypothesis, MARCHIAFAVA and BIGNAMI maintained that relapses at short intervals could best be explained by assuming that a certain number of the parasites survived quinine treatment and the action of the protective forces of the body. This statement however received no scientific support until ROSS and THOMSON by use of the "thick film" method were able to show that although the usual method of blood examination might, and very frequently did, fail to show parasites in the apyretic interval between relapses, in the thick films the organisms could be demonstrated in some cases during the entire interval. They also found that relapses so produced had no relation whatever to gametes or "resistant forms," but depended entirely on the degree to which the asexual parasites resisted quinine or the protective forces of the body. These facts are not accepted by CRAIG, DEADERICK and HENSON as explaining long interval relapses. BIGNAMI, however, after a careful examination of the claims made for a "resting stage" of the parasite, concludes that there is no basis for such claims. He argues from the view-point of analogy and biology that relapse is due to the persistence of the asexual cycle. The asexual parasites become immune either to the effect of quinine or to the protective forces of the body, just as EHRLICH has shown occurs in the case of trypanosomes with regard to atoxyl

and other drugs. He concludes that this immunity whether against quinine or against the protective forces of the body, will explain any case of relapse, whether at long or short intervals.

The author shows in detail how the known facts regarding relapse can best be accounted for by the second hypothesis. Parthenogenesis will not explain why triple quartan and double tertian infections relapse as such. It has been shown that the gametes do not live indefinitely. Relapses have been recorded after an interval of two years and there are certainly no facts to show that a gamete can persist for that length of time. The author believes that CRAIG's so-called "conjugation" forms of parasites are forms of developing crescents. The forms described by CRAIG undoubtedly exist but he has misinterpreted their meaning. There is a widespread belief that there are two kinds of relapses—one due to a simple renewal of vitality at short intervals, without further change of the asexual generation; and the other to a renewal of vitality at long intervals of some "resistant" or "latent" form, derived from the asexual cycle. The author however believes that there is but one primary cause of relapse, a renewal of vitality on the part of the asexual cycle, and that this is true, whether relapse takes place a week or a year after the original infection has subsided. The asexual cycle can persist over long periods, even two years, in the human host, though at times in numbers too small to be detected. This assumption seems quite reasonable, since other protozoa such as *Leishmania*, *Trypanosoma* and *Entamoeba* can also live in the human host for years. These infections are apparently maintained solely by the persistence of organisms that correspond to the asexual cycle of *Plasmodium*. CALKINS and WOODRUFF have shown that free living protozoa such as *Paramoecium* can reproduce themselves for many generations (over 3,000) without necessity for parthenogenesis or for conjugation. There is no reason why the asexual cycle of the malarial parasite should not persist for a time equal to the longest recorded interval between a primary infection and a relapse, viz. about two years. Malarial parasites of the asexual cycle may become immune to the protective forces of the body and ROSS and THOMSON, BIGNAMI, MOLLOY and others have shown that they may also develop a considerable resistance to quinine. The author has observed that the asexual parasites can be found in the spleen and bone marrow long after they have apparently disappeared from the peripheral blood. Possibly in these organs they are more able to resist the action of quinine.

It has been found from the large clinical experience obtained in Ancon Hospital that fewest relapses follow the treatment instituted by DEEKS, viz. 15 grains of quinine thrice daily by mouth for two to three weeks. This treatment resulted in fewer relapses than the previous treatment of 10 grains thrice daily. Treatment by intravenous injection is apparently the most certain of all. [This is an important paper and those interested should consult the original.]

BATES (John Pelham). **A Review of a Clinical Study of Malarial Fever in Panama.**—*Jl. Trop. Med. & Hyg.* 1913. June 16. Vol. 16. No. 12. pp. 177-184.

This is a continuation of the paper summarised in this *Bulletin*, Vol. 2, p. 150, and deals with the symptoms and differential diagnosis of malarial fever.

The author believes that there are two species of malignant tertian parasites to which he applies the term *Plasmodium falciparum subtertianum* and *P. quotidianum*. He does not believe that there is an nonpigmented form of the latter. *P. falciparum subtertianum* shows pigment early in its development. The pigment is coarse and granular, rather dark in colour, nearly always remains more or less aggregated, and occupies usually an eccentric position in the cytoplasm. The parasite does not attain the full size of its erythrocyte host; and in many instances shrinks and distorts the red blood cell to quite a considerable degree, producing the so-called "brassy bodies." *P. quotidianum* shows pigment late in its development. The pigment is in small granules, brownish in colour, and is usually disseminated throughout the cytoplasm until just before sporulation occurs, when it becomes aggregated about the centre of the organism. The parasite attains nearly or quite the full size of its erythrocyte host.

He describes at length the various signs and symptoms met with in malignant tertian malaria. He states that albuminuria is quite a common feature in malaria. Of 200 cases he found it in 42 per cent. In pernicious cases albumin and casts of various kinds are quite common. In all grave pernicious fevers hæmoglobin can be found in the urine if Brem's test be employed. Algid and choleraic types of pernicious malaria have been extremely rare in Panama. The author has seen one algid case, and one case of the choleraic form has been reported. He believes it is likely that these forms of malaria are due to a concomitant attack of heat-exhaustion as the symptoms are very like those of the latter disease. He does not believe that there is a true dysenteric type of malaria and maintains that the so-called dysenteric forms of malaria are simply cases of malaria complicated with true dysentery. Regarding the diagnosis of malaria he states that a fever which continues for more than five days unchecked by quinine is not malaria.

D. T.

ATKINSON (J. M.). **A Clinical Test for Malarial Fever.**—*Lancet.* 1913. June 28. pp. 1802-1803.

The author draws attention to SCHLESINGER's reaction for demonstrating the presence of urobilin in patients suffering from malarial fever. The test is as follows:—

Fill a test tube one-third full with unfiltered urine and add an equal quantity of Schlesinger's solution (zinc acetate one part, alcohol ten parts) which has previously been well shaken; then drop in a few drops of weak solution of tincture of iodine—this

accelerates the reaction. The mixture is then filtered and, if urobilin be present, shows a more or less distinct fluorescence.

The reaction is met with in the urine of patients suffering from all types of malaria and in cases of malignant malaria it may even be obtained after diluting the urine with 200 parts of water. A healthy urine does not give the reaction as it contains only the slightest amount of urobilin, but other diseases such as cirrhosis of the liver, abscess, and many infectious maladies also exhibit it so that the test cannot be described as specific. On the contrary, as the author points out, its absence speaks strongly against acute malarial fever, a fact of great value when a quick diagnosis has to be made.

G. C. L.

HAMMOND (F. A. L.). **Malarial Gangrene.** [Mirror.]—*Indian Med. Gaz.* 1913. June. Vol. 48. No. 6. p. 228.

The author describes two very interesting cases of moist gangrene occurring in native patients suffering from severe attacks of malignant tertian malaria. On admission to the hospital both patients were in a state of collapse with subnormal temperature.

Case 1, Hindoo male, aged 19, showed a very heavy infection of malignant tertian malaria. One in every three corpuscles contained parasites. Stimulants were administered and quinine grs. 10 was injected hypodermically each day. On the 5th day it was noticed that the skin over both calves was becoming dusky in patches, which spread rapidly over the whole surface of the leg. In spite of all precautions moist gangrene became three days later fully established in both lower extremities. The right leg had to be amputated at the knee. The left foot subsequently sloughed. There was no history of syphilis in this patient. He eventually recovered.

Case 2, Mahommedan, male, aged 25, had had fever for 8 days before admission. His pulse was imperceptible, and the whole body was cold and clammy. In spite of every effort to improve his vitality by brandy, adrenalin, strychnine, digitalis, and the intravenous administration of quinine, he developed gangrene of all four extremities, which rapidly became moist and septic. He died fourteen days after admission, having developed oedema of the lungs.

D. T.

NICHOLLS (Lucius). **Cirrhosis of the Liver of Malarial Origin.**—*Jl. Trop. Med. & Hyg.* 1913. June 2. Vol. 16. No. 11. pp. 164-165. With 1 plate.

The author states that cirrhosis of the liver is a common condition in many tropical countries, and numerous authorities have asserted that some cases are caused by repeated attacks of malaria; this however has been disputed, and at the present time

there appears to exist much doubt that malaria is ever a cause of the condition. In the author's opinion a type of monolobular biliary cirrhosis does arise from repeated attacks of malaria. The production of this cirrhosis depends upon the presence of adhesions and enlarged glands in the transverse fissure of the liver and thickening of the fibrous capsule. The sequence of changes appears to be:—1. The formation of adhesions, and enlargement of lymphatic glands, which press upon the bile-ducts in the transverse fissure; (2) The swelling of the liver at each attack of malaria; (3) As the adhesions and perihepatitis increase, the organ is held as in a vice, and the hepatic enlargement must take place against considerable pressure; (4) There is therefore increased internal pressure in the bile ducts and gall bladder; (5) Malarial toxins are formed in the liver, and these being excreted under unusual pressure along the bile ducts, result in the formation of fibrous tissue around the latter.

The author therefore believes that malarial cirrhosis is primarily due to mechanical causes arising from adhesions in the transverse fissure of the liver and a general thickening of the fibrous capsule of the organ.

D. T.

DE VILLA (S.). **Tremore essenziale da Malaria.** [Tremor due to Malarial Infection.]—*Gaz. Internaz. di Med., Chir., Igiene.* 1913. May 10. No. 19. pp. 443-444.

The author discusses numerous disturbances of the nervous system due to malaria. He describes in detail the case of an infant of 15 months who had previously suffered from that disease. The patient was cured by quinine, but 5 days after the last attack of hyperpyrexia developed a general tremor. This consisted of very rapid rhythmic and uniform oscillations which continued during sleep. No malarial parasites were found in the blood at this stage.

D. T.

CANTIERI (Collatino). **Sul particolare Decorso Termico della Pneumonia Crupale in molti Soggetti Malarici (Pneumonia Remittente-intermittente dei Malarici).** [On the Pyrexial Course of Croupous Pneumonia in Malarial Subjects. Remitting Intermittent Pneumonia of Malarial Subjects.]—*La Clinica Medica Italiana.* 1913. Jan. Vol. 52. No. 1. pp. 30-51.

Croupous pneumonia when occurring in a malarial patient follows a special pyrexial course with intermittences and remittances varying in the different cases. At the onset the disease frequently simulates malarial fever; quinine however has no effect.

D. T.

## LABORATORY AND EXPERIMENTAL.

- i. ZIEMANN (Hans). On the Culture of Malarial Parasites and *Piroplasma canis*.—*Trans. Soc. Trop. Med. & Hyg.* 1913. May. Vol. 6. No. 6. pp. 220-227.
- ii. Über die Kultur der Malariaparasiten und der Piroplasmen (*Piroplasma canis*) in vitro.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. June. Vol. 17. No. 11. pp. 361-391. With 2 coloured plates [issued in No. 13] and 2 curves.

i. In the cultivation of malarial parasites the author used 5 c.c. of the patient's blood to which was added 1/10 c.c. of a 50 per cent. solution of dextrose. The blood was defibrinated and the leucocytes removed by centrifuging, the top layer of serum and the bottom layer of corpuscles being transferred to another tube. He obtained the best results by using inactivated dextrose-ascitic serum. The serum was inactivated by heating to 43° C. for one hour. The malarial parasites showed in culture exactly the same morphological and biological development as in the human organism. In the culture tube the development of the benign tertian parasites took place in about 34 hours at a temperature of 39.5° C. while *Plasmodium falciparum* underwent its full development in 40-48 hours at a temperature of 37° C.; but occasional differences in the time required for their full development occurred.

In the culture tubes he found, in addition to normal parasites, forms which contained vacuoles and in which the chromatin was broken up and scattered. Such forms were never seen in the peripheral blood, though they reminded one of the degenerated quinine forms sometimes seen. In the cultures he noticed that the leucocytes ingested the degenerating and dying forms of the parasites. He was unable to observe any signs of conjugation of young schizonts as described by MANNABERG and later by CRAIG and he found no evidence of the occurrence of parthenogenesis or the formation of ookinets. The young merozoites apparently remained extra-corpuseular in the blood plasma only for a very short time after sporulation occurred. He was unable to notice that the parasites wandered from infected red cells to non-infected ones, as maintained by Mary Rowley LAWSON. He does not share the belief of BASS that it is possible to cultivate malarial parasites ad infinitum, without sexual phases intervening.

The author has also attempted the cultivation of *Piroplasma canis* by BASS's method, and succeeded after several experiments. He found that the best growth of this parasite was obtained when the blood of dogs containing few parasites was used. The growth was less successful when the blood of heavily infected dogs was employed. If the spleen is removed three or four days before taking the blood, the cultivation is invariably successful. His technique was as follows:—To 5 c.c. of defibrinated dogs' blood add 1/10 c.c. of 50 per cent. glucose solution and 0.15 c.c. of a 2 per cent. solution of sodium citrate. It is better to inactivate the serum and the leucocytes should be removed. The best

growth took place at a temperature of 37° C., but the parasites were able to grow at room temperature and also at 40° C. Sporulating forms of this parasite with more than 4 merozoites are very rarely seen in the peripheral blood of the dog, but in the culture tubes forms with 16 and more merozoites were frequently seen. The cultures remained virulent for 5 and even 6 days. He was able to obtain subcultures from a culture four days' old. After some days the parasites show signs of degeneration in the culture tubes. They lose their typical pear shape, and become rounded. The protoplasm takes on a darker stain while the colour of the chromatin becomes more faint.

ii. This article is almost identical with that summarised above except that in addition a short history is given of the previous attempts made to cultivate malarial parasites in vitro up to the time when this was first successfully accomplished by BASS in 1912. The number of spores obtained by the author in his cultures of *Plasmodium falciparum* was 14 to 18, most frequently 16. He gives a full bibliography of the cultivation of malarial parasites and of *Piroplasma canis*.

D. T.

THOMSON (John Gordon). **A Demonstration on the Cultivation of the Malarial Parasites** (*Plasmodium falciparum* and *Plasmodium vivax*).—*Trans. Soc. Trop. Med. & Hyg.* 1913. May. Vol. 6. No. 6. pp. 216-219. With 1 plate.

The author gives the following conclusions regarding the cultivation of these parasites:—

1. "*Plasmodium falciparum* and *Plasmodium vivax* are capable of being cultivated up to the stage of sporulation, for at least one generation, and we have obtained evidence that *Plasmodium falciparum* may develop for several generations (at least three) in the original culture tube, without transplanting to a new medium.

2. "In some cultures the growth was rapid, in others much slower; this is explained by different factors:—(a) The age of the parasite when introduced into the culture tubes. (b) The temperature of the incubator. (c) The amount of glucose used. (d) The influence of previous administration of quinine to the patient.

"With respect to (d), we have noted that on several occasions sporulation in the case of malignant tertian fever was not completed when the patient had been treated with quinine, the maximum number of spores in cultivations prepared from these being about ten. In several cases the parasites refused to develop, and we attributed this to the fact that quinine had been administered before the blood was drawn for cultivation.

3. "The cultures of benign tertian differed from those of malignant tertian in that there was no tendency to clumping of parasites in the former, either before or during sporulation.

4. "This difference appears to us to explain in a satisfactory manner why only young forms of malignant tertian are found in the peripheral blood, as the clumping tendency of the larger forms causes them to be arrested in the finer capillaries of the internal organs. It also explains the tendency to pernicious symptoms, such as coma, in malignant tertian malaria. All stages of the benign tertian parasites are found in the peripheral blood, and there are seldom pernicious symptoms, because there is no tendency to clumping.

5. "The malignant tertian parasite (*P. falciparum*) is capable of producing, in maximum segmentation, 32 spores. On the other hand, benign tertian (*P. vivax*) produces, as a rule, during maximum segmentation, 16 spores; sometimes more may be produced, but the number is never 32.



6. "The pigment of *P. falciparum* collects into a definite, circular, and very compact mass early in the growth of the parasite. On the other hand, during the growth of *P. vivax*, the pigment remains scattered in definite granules throughout the body of the parasite, till just before segmentation, when it collects into a loose mass of granules in the centre of the full-grown *Plasmodium*."

D. T.

GURKO (A. G.) & HAMBURGER (J.). Zur Frage über die Kultur des Plasmodiums der tropischen Malaria nach Bass und Johns.—Vorläufige Mitteilung. [On the Culture of the Plasmodium of Malaria Tropica according to Bass and Johns.—Preliminary Note.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. May 20. Vol. 74. No. 2. pp. 248-252.

The authors give a resumé of the attempts made by various workers to preserve or cultivate the malarial parasite, and a full account of BASS's method.

Of the authors' three experiments the first resulted in the successful growth of three generations, whilst the other two proved negative. The first was made with the blood of a boy who had never received quinine or any other treatment, and containing rings and crescents of 'malaria tropica.' The second experiment was made with blood from the same patient after 8 days' quinzation and containing crescents but no ring forms. The blood for the third experiment contained crescents as well as a few rings and was taken from a boy who had had quinine.

The authors state that their observations are too few to allow of any conclusions as to the cause of the negative results in the last two experiments. Further observations are stated to be in progress.

D. T.

BILLET (A.). Action de la Quinine sur les Hématozoaires du Paludisme.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 336-339.

Billet agrees with RIEUX (this *Bulletin*, Vol. 2, p. 8) regarding the action of quinine salts on the various stages of the malarial parasite in the human blood. The author used in his experiments the formate of quinine, known under the name of quinoform. He made observations on five cases of benign tertian, one case of malignant tertian, and one case of quartan malaria. The dose of quinoform, given by mouth in a cachet, was 0.5 grammes and in two cases only 0.3 grammes. The blood was then examined hourly.

These isolated doses produced the following results:—

(1) Disintegration of the protoplasm of the benign tertian schizonts occurred sometimes as early as one hour after the administration of the drug. It was very marked after 5 hours, and the fragmentation was complete in 10 to 12 hours. The sexual parasites or gametes resisted the action of the quinine for 24 to 48 hours or longer. It is even probable that the gametes

can develop to maturity, completely resisting the action of the drug.

(2) The different phases in the degeneration of the parasites due to quinine are as follows:—(a) retraction of the protoplasmic processes; (b) disappearance of the nutritive vesicle; (c) progressive fragmentation of the protoplasm; the various portions contain pigment granules and are scattered more or less over the entire area of the corpuscle; (d) isolation and prolonged persistence of the nuclear chromatin which seems to be the most resistant part of the parasite; (e) finally the protoplasmic debris with the pigment is shed into the blood plasma and becomes ingested by the large mononuclear leucocytes. The enlarged, decolorised and deformed red cells can be seen, emptied of their parasites but still showing Schüffner's granules and sometimes masses of pigment.

(3) The action of quinine is more powerful upon the young parasites than upon those which are more fully developed; hence the best time to give the drug, in order to get the maximum destructive effect, is immediately after the liberation of spores, or just about the end of the febrile attack.

(4) The effect of quinine was equally rapid in the case of malignant tertian.

(5) The quartan parasites appeared to be more resistant than the benign tertian, but they eventually underwent the same phases of degeneration.

D. T.

#### PROPHYLAXIS.

Ross (Ronald). **Malaria Prevention in Greece.** [Correspondence.] —*Brit. Med. J.* 1913. May 31. p. 1186.

The author visited Greece seven years ago. At that time the British employees of the Lake Copais Co., suffered much from malaria. He advised that the neighbouring collections of water should be kept free from weeds and periodically oiled and that the houses should be rendered mosquito proof. In 1913 he again visited the locality and found that there had been no case of malaria among these employees for some years past, his recommendations having been carried out thoroughly. In the neighbouring Greek village of Moulki much quinine has been distributed by the Grecian Government, especially to the children. In 1906 the author found splenomegaly in 56 per cent. of the children. This year he found it only in 28 per cent. of the children examined. He considers however that the spleen rate has been reduced 50 per cent., since in the later examination he included spleens showing very slight enlargement. Moreover the children and the villagers in general looked in every way much healthier than they did on his former visit. The pools around the village still remain, although they are oiled periodically. He believes that it would be cheapest and best in the end to abolish these pools. Shortly after his first visit, the Liverpool School of Tropical Medicine raised a subscription of several hundreds of

pounds for the Greek Antimalarial League. This league persuaded the Greek Government to make quinine a state monopoly in order to avoid expense and adulteration. The League is doing excellent work, but the Greek Government only gives it 5,000 drachmae (francs) a year. On the other hand the Government is making profit out of the sale of quinine, amounting to 83,000 drachmae in 1911. The author urges that this money should be used for malaria prevention.

D. T.

MEREU (Francesco). *La Malaria in Nurra. Condizioni Sociali, Economiche ed Igieniche.* [Malaria in Nurra, Sardinia. Social, Economic, and Hygienic Conditions.]—*Malaria e Malat. d. Paesi Caldi.* 1913. Apr.-May. Vol. 4. No. 3. pp. 182-188.

Malaria plays an important role in the death-rate of Nurra. The malarial season, from July to November, is at its height in September. Aestivo-autumnal fever is more frequent than tertian; quartan is entirely absent.

D. T.

- i. BRIGNONE (Emiliano). *La Propaganda e Profilassi Antimalarica nelle Scuole comunali di Terranova Monferrato durante l'Anno 1912. Relazione a S.E. il Ministro dell' Istruzione.* [Antimalarial Propaganda and Prophylaxis in the Elementary Schools of Terranova Monferrato in 1912. Report to the Minister of Education.]—*La Propaganda Antimalarica.* 1913. June. Vol. 6. No. 3. pp. 57-68.
- ii. MALTESE (Paolo). *Educazione Antimalarica e Profilassi Antimalarica Scolastica nella Provincia di Trapani. Relazione a S.E. il Ministro della Pubblica Istruzione.*—*Ibid.* Apr. No. 2. pp. 36-45.
- iii. SERGI (Antonio). *La Profilassi scolastica antimalarica nel 1912 in Palizzi Marina.*—*Ibid.* June. No. 3. p. 69.

i. Climatic conditions were most favourable to the development of mosquitoes during this year; *A. maculipennis* and *A. bifurcatus* were very numerous. Realizing the danger of a severe recrudescence of malarial fever, the author organised a system of rigid vigilance and prophylaxis.

Public instruction was given; more especially instruction to the children at the schools. The author explains at length the advantages of scholastic instruction over all other forms of anti-malarial propaganda. State quinine was given to the school children on alternate week days from March to June. All these measures were scrupulously carried out and the results according to the author were brilliant.

ii. This Report is drawn up on the same lines as that by Brignone; also here the results obtained were excellent. A series of lectures on the malarial problem had been given to the school teachers.

iii. The same excellent results were obtained. Among 68 school children who received their quotidian dose of state quinine, only one slight case of benign tertian developed after the first days of the quinization, in a child who had had malaria previously. No further cases developed during the two months of this treatment. During the same period 57 cases of malaria were reported amongst the population of 2,000 (*i.e.*, 171 per thousand per annum).

D. T.

SKELTON (D. S.). **Report on the Measures Necessary to reduce Malarial Fever in Zanzibar.** — Report dated Health Office, Zanzibar, Feb. 15, 1913. Received in Colonial Office Apr. 30, 1913.

The author states that malaria is responsible for about 175 deaths a year in Zanzibar town. Since the mortality rate in malaria is very low, this number indicates that the disease must be very prevalent. The measures which he considers necessary to reduce the malaria rate are excellent; among others he suggests that a mosquito brigade should be formed consisting of four mosquito inspectors, twelve workers, and one inspector for swamps. The estimated cost of this would be about £400 a year. It is hoped that the Colonial Office will approve and adopt his suggestions.

D. T.

#### MONKEY MALARIA.

BOUILLIEZ (M.). **Nouvelles Recherches expérimentales sur un Plasmodium des Singes.** — *Compt. Rend. Soc. Biol.* 1913. May 23. Vol. 74. No. 18. pp. 1070-1072.

The author has inoculated successfully several species of monkeys with *Plasmodium inui*. In most of these the infection was chronic, but one monkey (*Cercopithecus callitrichus*) had a very acute attack to which it succumbed, twelve days after inoculation. This monkey moreover developed haemoglobinuria just before it died. In two cases splenectomy was performed. In the first case it appeared to cause a more prolonged exacerbation of the disease than usual. In the second case it resulted in a very acute attack with increase in the number of parasites and death in six days. He was unable to find any morphological change in the parasites such as was described by BOUNIOL as the result of splenectomy (see this *Bulletin*, Vol. 2, p. 157). Two monkeys, of 900 and 1,300 grammes respectively, were inoculated with the parasites and at the same time with 7 centigrammes and 12·5 centigrammes of quinine. The quinine was injected into one thigh, the inoculation into the other. Neither contracted the disease. The author concludes that it had a prophylactic effect. He was unable however to find that quinine even in very large doses had any curative effect in monkeys. Two animals died of the disease in spite of the quinine treatment. An attempt was

made to cultivate the parasites according to Bass's method. No success was obtained by incubating at a temperature of 40° C., but at a temperature of 22° C. there was apparently an increase in the number of parasites after six days, and numerous rosette forms appeared.

D. T.

### BLACKWATER FEVER.

**SOREL.** *Traitement de la Fièvre Biliéuse Hémoglobininurique par les Injections et Lavages de Solutions Sucrées.* [Clinique d'Outre-Mer.]—*Ann. d'Hyg. et Méd. Coloniales.* 1913. Jan.-Feb.-Mar. Vol. 16. No. 1. pp. 194-199.

During the last two years the author has treated six cases of blackwater fever at Bassam (West Africa). All recovered. He maintains that this disease, whatever the cause, is essentially a toxic condition. It is therefore good practice to keep the kidneys acting by means of hot drinks and saline injections; but this is insufficient, for in many cases the patient succumbs to an intoxication of the nervous system, even though the action of the kidneys is well maintained. This nerve intoxication is indicated by the feeble irregular pulse, by the state of semi-delirium and finally death from syncope. FLEIG, ARROUS and JEANBRAU have demonstrated that intravenous injections of glucose, lactose, or mannite solutions have a powerful tonic action on the heart and a specific action on the nerve centres in that they cause a rapid elimination of toxins such as occur after anaesthesia in surgical operations; moreover they have injected intravenously as much as 1,300 c.c. of these solutions without producing any bad effects on the kidneys. The author on these grounds has employed sugar solutions in the treatment of blackwater fever in six cases.

Case I. The patient suffered from nephritis and during his blackwater attack passed very little urine in spite of hot drinks and saline injections. Later he developed complete suppression. The author then injected 300 c.c. of an isotonic lactose solution, and 12 hours later 250 c.c. The patient passed urine freely in two hours after the first injection. After 12 hours Sorel administered another injection of 350 c.c. lactose and also washed out the bowel with a litre of the same solution. The patient improved rapidly and recovered in a very satisfactory manner.

Details are given of the remaining five cases which were treated successfully in a similar manner. Glucose solutions appeared to be as beneficial as the lactose solutions. These solutions were made in the following strengths: crystallised lactose 92.5 grammes, water 1,000 c.c.; crystallised glucose 47 grammes, water 1,000 c.c. The author suggests that on three consecutive days a subcutaneous injection of 250 c.c. should be given in the morning, followed by an injection per rectum of 700 to 800 c.c. in the evening. In a very grave case he obtained most beneficial results after an intravenous injection of 250 c.c. of the glucose solution.

D. Thomson.

LOVELACE (Carl). **The Etiology and Treatment of Hemoglobinuric Fever. A Report of Five Hundred and Fourteen Cases.**—*Arch. Internal Med.* 1913. June 15. Vol. 11. No. 6. pp. 674-684.

This paper is based on 514 cases of blackwater fever treated by American physicians in the hospital of the Madeira-Mamore Railway Company, Porto Velho, Brazil, between January 1, 1908, and November 27, 1912. [If all these cases were really blackwater fever it indicates a very high degree of infection in those parts.] The study brings out, what has so often been brought out before, that malarial infection stands in a direct causal relationship to blackwater fever, that blackwater fever is not due to any particular species of malarial parasite, that quinine in large or small doses may be an antecedent of the haemoglobinuria, that this drug should not be given during the attack, that normal saline solution should be given to keep up the blood pressure, and that the prophylaxis of malaria is the prophylaxis of blackwater fever.

G. C. Low.

LOW (George C.) & WENYON (C. M.). **Cell Inclusions in the Leucocytes of Blackwater Fever and other Tropical Diseases.**—*Jl. Trop. Med. & Hyg.* 1913. June 2. Vol. 16. No. 11. pp. 161-163. With 1 coloured plate and 2 text-figs.

The authors have searched for cell inclusions in the blood in various tropical diseases, viz.: malaria (recent and chronic), blackwater fever, trypanosomiasis, sprue, filariasis, and anaemias of various kinds. Inclusions occurred chiefly in the cells of the large endothelial type. They believe that the largest inclusions are derived from ingested normoblastic nuclei. The medium sized inclusions would seem in many cases to originate in the nuclei of the cells themselves, but in other instances might result from the ingestion of parasites containing chromatin (trypanosomes, leishmania, malaria, etc.). They might also be the products of disintegration of ingested nuclei, such as the nuclei of lymphocytes or normoblasts (nucleated red cells). In the coloured plate accompanying their article there are depicted various types of cell inclusions; among these is a remarkable display of inclusions in the large endothelial cells from the peritoneal exudate of a mouse inoculated with *Leishmania tropica*. These represent the nuclei of various ingested cells in different stages of disintegration. In a severe fatal case of blackwater fever they failed to find large endothelial cells or inclusions. So far as they can judge from the varying origin and occurrence of these inclusions it does not seem legitimate at present to associate them with any particular disease, and in their opinion there seems to be no possibility of their being parasitic in nature. They believe that the bodies described recently by COLES in a case of blackwater fever blood resemble artefacts more closely than anything else, and they certainly do not look upon them as parasites. Apart from the cell inclusions in the leucocytes therefore, and these would not appear to be parasitic in origin,

no other appearances resembling protozoal parasites, so far as they know, have been seen in the blood of blackwater fever by the many investigators who have studied the disease.

D. T.

BOOGHER (Leland). **Malarial Hematuria.**—*New York Med. Jl.* 1913. June 21. Vol. 47. No. 25. pp. 1291-1293.

Three cases of "malarial haematuria" are described. In all of these the urine contained large numbers of red blood corpuscles in which, the author states, he saw malarial plasmodia. One of the cases had lived in a malarial district all his life but had enjoyed good health up to the year previous to his attack. The other two patients were said to have suffered from malaria previously. [The author does not bring forward any definite proof in the history of his cases that these haematurias were really due to malaria—they were evidently not cases of blackwater fever with which disease he has confused haematuria.]

G. C. L.

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## SLEEPING SICKNESS.

## TRYPANOSOMIASIS IN NYASALAND AND RHODESIA.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. **Morphology of Various Strains of the Trypanosome causing Disease in Man in Nyasaland.—The Wild-game Strain.**—*Proc. Roy. Soc.* 1913. June 12. Series B. Vol. 86. No. B 589. pp. 394-407. With 7 charts.

The wild-game strains were isolated by injecting blood of antelope into susceptible animals—goat, monkey and dog—and from these other animals were inoculated. Trypanosomes from the following species were studied—reedbuck, waterbuck, oribi and hartebeeste (2).

The results obtained by measuring 500 individuals of each of the five strains in rats are set forth in tables and charts.

Unlike the curves of the Human strains those obtained from the wild-game strains are all remarkably alike, and there can be little doubt that the same species of trypanosome is being dealt with in all five strains. The wild-game curves resemble those of the Human strains II, IV, and V described in a former paper (see this *Bulletin*, Vol. 1, p. 659) and also those found by KINGHORN and YORKE in the Luangwa Valley (*Sleeping Sickness Bulletin*, Vol. 4, p. 170).

The percentage of posterior nuclear forms found among the short and stumpy varieties of the trypanosomes of the five wild-game strains was 8·4, 30·7, 30·3, 28·3 and 33·4 respectively.

The following are the conclusions:—

1. The five Wild-game strains resemble each other closely, and all belong to the same species of trypanosome.
2. The Wild-game strains and the Human strains, although they differ to some extent, also belong to the same species.
3. This species is *T. rhodesiense* (Stephens and Fantham).
4. There is some reason for the belief that *T. rhodesiense* and *T. brucei* (Plimmer and Bradford) are one and the same species.

W. Yorke.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. **Morphology of Various Strains of the Trypanosome causing Disease in Man in Nyasaland. — The Wild *Glossina morsitans* Strain.**—*Proc. Roy. Soc.* 1913: June 12. Series B. Vol. 86. No. B 589. pp. 408-421. With 7 charts.

The strains were obtained by bringing wild tsetse flies (*Glossina morsitans*) to the laboratory from the neighbouring "fly country" and at once allowing them to feed on healthy animals. The first strain was obtained by feeding the flies on a monkey, the remaining four by feeding on dogs. As soon as the healthy animal was found to be infected other animals were



inoculated from it. As in the case of the wild-game strains, only trypanosomes from a single rat were used for purposes of measurement and comparison.

The results obtained by measuring 500 individuals of each of the five strains are given in tables and charts.

The curves of the wild-game strains and the wild *Glossina morsitans* strains are so similar that there can be little doubt that the same species of trypanosome is being dealt with. The Human strain differs so much, that "the suspicion must present itself that in some way more than one species is being dealt with." The three Human strains which differ most from the wild-game and wild *Glossina morsitans* type are STEPHENS and FANTHAM's case of Armstrong, and strains I and III (see this *Bulletin*, Vol. 1, p. 659). On examining these three strains, however, from every possible point of view, nothing except the difference in the type of the curve can be found to justify this suspicion.

The following are the conclusions:—

1. The five Wild *Glossina morsitans* strains resemble each other closely, and all belong to the same species of trypanosome.
2. The Wild *Glossina morsitans* strain, the Human strain, and the Wild-game strain, belong to the same species.
3. This species is *T. rhodesiense* (Stephens and Fantham).
4. It is probable that *T. rhodesiense* and *T. brucei* (Plimmer and Bradford) are identical.

[The work of STEPHENS and BLACKLOCK (see this *Bulletin*, Vol. 1, p. 662) showed that *T. brucei* (PLIMMER and BRADFORD) is a monomorphic trypanosome and easily distinguishable from the polymorphic *T. rhodesiense*. LAVERAN in a more recent paper (see this *Bulletin*, Vol. 2, p. 137) supports the conclusions of STEPHENS and BLACKLOCK.]

W. Y.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. Infectivity of *Glossina morsitans* in Nyasaland.—*Proc. Roy. Soc.* 1913. June 12. Series B. Vol. 86. No. B 589. pp. 422-426.

This paper records the results of feeding 10,081 wild *Glossina morsitans*, caught in the "Proclaimed Area" of Nyasaland, on monkeys, dogs and goats. Each cage of flies was fed on three healthy animals—the first day on a monkey, the second on a dog, and the third on a goat. To ensure, as far as possible, that each animal was fed on by every fly, the flies were fed nine times—three times on each animal.

The results obtained by feeding these flies, in 56 batches, are given in a table, and show that four species of trypanosomes, viz., *T. brucei* vel *rhodesiense*, *T. pecorum*, *T. simiae* and *T. caprae*, are carried by *Glossina morsitans* in this district.

Ten thousand flies gave rise to 135 infections and, assuming that no fly was infective with more than one species of trypanosome, 13.5 per 1,000 flies are infective with one or other of the disease-producing trypanosomes of this district.

Table III.—The Proportion per 1,000 Tsetse Flies, caught in the "Sleeping Sickness" Area of Nyasaland, found to be infective with Pathogenic Trypanosomes.

<i>T. Brucei</i> vel <i>rhodesiense</i> .	<i>T. pecorum</i> .	<i>T. simiae</i> .	<i>T. caprae</i> .
Per 1,000. 2	Per 1,000. 4·6	Per 1,000. 3·4	Per 1,000. 3·5

Infective flies occur all the year round and are just as numerous during one season as another. No experiments were carried out during July and August.

The following are the conclusions:—

1. The tsetse flies (*Glossina morsitans*) caught in the "fly-country" near Kasu are infected with four species of disease-producing trypanosomes—*T. brucei* vel *rhodesiense*, *T. pecorum*, *T. simiae*, and *T. caprae*.

2. The proportion of infective flies is 13·5 per 1,000.

3. The proportion of flies infective with *T. brucei* vel *rhodesiense*, the cause of the Human Trypanosome Disease of Nyasaland, is 2 per 1,000.

4. The flies are found infective all the year round.

5. To prevent the infection of tsetse flies it is proposed that the experiment should be tried of destroying all the wild game in the "Proclaimed Area" of Nyasaland.

[These results confirm and extend those of KINGHORN and YORKE in the Luangwa Valley of Northern Rhodesia (*Sleeping Sickness Bulletin*, Vol. 4, p. 321).]

W. Y.

KINGHORN (Allan), YORKE (Warrington), & LLOYD (Llewellyn).

Final Report of the Luangwa Sleeping Sickness Commission of the British South Africa Company 1911-1912.—*Ann. Trop. Med. & Parasit.* 1913. June 10. Vol. 7. No. 2. pp. 183-302. With 12 plates.

Most of the contents of this Report have been already published and have been noticed in this and in the *Sleeping Sickness Bulletin*.\* The matter is here somewhat differently arranged.

Section I by KINGHORN and YORKE deals with the human trypanosome. There are considered the distribution of the disease; the clinical features; the identity of the trypanosome with *T. rhodesiense*; the transmission of the trypanosome in the valley and on the plateau respectively; the influence of meteorological conditions on the development of the trypanosome in *Glossina morsitans*; the reservoir of the trypanosome; the occurrence of the trypanosome in *Glossina morsitans* in nature; the identity of the game and fly strains with the human strain of *T. rhodesiense*. The summary is as follows:—

"1. The human trypanosome (*T. rhodesiense*) is distributed widely throughout South Central Africa.

"2. There is no essential difference between the clinical manifestations of the disease in man caused by *T. rhodesiense* and that due to *T. gambiense*, except possibly the greater virulence of the former.

\* See *Sleeping Sickness Bulletin* Vol. 4, pp. 165, 170, 235, 315, 321, and *Tropical Diseases Bulletin* Vol. 1, pp. 43, 126, 268, 276, 516.

"3. *T. rhodesiense* is transmitted in Rhodesia by *Glossina morsitans*.

"4. Approximately 3.5 per cent. of the flies may become permanently infected and capable of transmitting the virus.

"5. The period which elapses between the infecting feed of the flies and the date on which they become infective varies from eleven to twenty-five days in the Luangwa Valley.

"6. Attempts carried out at laboratory temperature on the Congo-Zambesi plateau during the cold season to transmit the human trypanosome by means of *Glossina morsitans* were invariably unsuccessful in spite of the fact that 680 flies were used in these experiments.

"7. The developmental cycle of *T. rhodesiense* in *Glossina morsitans* is to a marked degree influenced by the temperature to which the flies are subjected. High temperatures (75-85° F.) favour the development of the parasite, whilst low temperatures (60°-70° F.) are unfavourable.

"8. The first portion of the developmental cycle can proceed at the lower temperatures, but for its completion the higher temperatures are essential.

"9. The parasites may persist in the fly at an incomplete stage of their development for at least sixty days under unfavourable climatic conditions.

"10. These observations afford an adequate explanation of the extremely long latent periods of trypanosomes in *Glossina* which have occasionally been observed by various workers.

"11. The relative humidity of the atmosphere has apparently no influence on the development of the trypanosome in *Glossina morsitans*.

"12. Mechanical transmission does not occur if a period of twenty-four hours has elapsed since the infecting meal.

"13. *Glossina morsitans*, in nature, has been found to transmit the human trypanosome.

"14. The chief reservoir of the human trypanosome is the antelope

"15. The results of examination for the human trypanosome of the blood of a large number of monkeys, wild rats and mice were invariably negative."

Section II, by the same authors, deals with the trypanosomes of game and domestic stock. The conclusions reached are as follows:—

"Trypanosomes are of frequent occurrence in game and domestic stock in North Eastern Rhodesia. As a conservative estimate the percentage of big game infected with trypanosomes pathogenic to man and domestic stock may at Nawalia (Luangwa Valley) be placed at 50, and at Ngoa (Congo-Zambesi watershed) at 35.

"At Nawalia six species of trypanosomes were isolated from game and domestic stock, viz., *T. rhodesiense*, *T. vivax*, *T. nanum*, *T. pecorum*, *T. montgomeryi*, and *T. multiforme*; whilst at Ngoa five species were found, viz., *T. rhodesiense*, *T. vivax*, *T. nanum*, *T. pecorum*, and *T. trachelaphi*.

"The results of examination of over 400 monkeys, wild rats and mice were invariably negative."

Section III deals with the trypanosomes found in wild *Glossina morsitans*. The summary is as follows:—

"*T. rhodesiense*, *T. ignotum* and *T. pecorum* are transmitted by *Glossina morsitans* in nature, and were obtained by feeding wild freshly-caught *Glossina morsitans* on healthy monkeys."

Section IV deals with the description of the trypanosomes found. All are figured in coloured plates. *T. multiforme* was isolated from a bushbuck at Nawalia (valley). It is stated that this parasite is at once distinguished from *T. pecaui* and *T. rhodesiense* by the absence of posterior nuclear forms and by its slight pathogenicity for laboratory animals. It closely resembles *T. gambiense*, from which it is not easily distinguished. A study of the biometric curve shows certain differences, and a comparison of the percentages of short, intermediate, and long forms shows that this parasite has very few intermediate forms (the percentage

is 12.5 against 23.1 for *T. gambiense*). The authors conclude that the parasite is a new species. The biometric curve is given, a table of measurements, and a table of pathogenicity.\*

Writing of *T. ignotum*, which, the authors say, is almost certainly identical with *T. simiae* of BRUCE, they note that three monkeys inoculated on the plateau with the valley strain of the parasite failed to become infected.

*T. tragelaphi* was found in blood films made from a sitatunga shot near Mpika; it bears a very close resemblance to *T. ingens*. Five specimens were seen. Being shorter and more slender than *T. ingens* it is described as a new species. The sitatunga lived in a large swamp at least fifteen miles from a *Glossina morsitans* area. It is suggested that leeches, which were found in enormous numbers, are the vectors of the parasite, more particularly as it is very like an amphibian trypanosome.

Section V dealing with the development of *T. rhodesiense* in *Glossina morsitans* is by KINGHORN, YORKE and LLOYD. The summary is as follows:—

"1. The salivary glands of all *Glossina morsitans* capable of transmitting *T. rhodesiense* are infected, and conversely without invasion of the salivary glands there is no infectivity of the fly.

"2. Invasion of the salivary glands is secondary to that of the intestine.

"3. The first portion of the developmental cycle of the trypanosome takes place in the gut. In order for its completion and for invasion of the salivary glands to occur, a relatively high mean temperature, 75°–85° F., is necessary.

"4. Invasion of the salivary glands was only found in flies infected with the human trypanosome, *T. rhodesiense*.

"5. The predominant type of the trypanosome in the intestine of infected *Glossina morsitans*—a large broad form—is quite different from that which predominates in the salivary glands, where the parasite resembles somewhat the short form seen in the blood of the vertebrate host.

"6. Both the intestinal forms and also those from the salivary glands of infective *Glossina morsitans* are virulent when inoculated into healthy animals."

A coloured plate shows five typical forms seen in the salivary glands and nine intestinal forms.

Section VI is the report of the Entomologist, Ll. LLOYD. Much of this has not previously been published. It is noted that in collections of *G. morsitans* the percentage of females was often as low as 2. Of the flies bred in the laboratory the pupation period varied from 21 days in a pupa exposed to a temperature slightly over 86° F. to 88 days for a pupa exposed to 62° F., but such extremely high and low temperatures had a deleterious effect on the pupae; in a large percentage the flies did not emerge. Details are given of places where pupae were discovered in nature, with many photographs. They were found in association with trees of eight different species and in holes in the earth; all the trees were either abnormal or injured. They were generally, though not always, hidden by a slight covering of earth or dead leaves. Of the 54 living pupae found 49, or 90 per cent., were so placed that they would be daily warmed by the sun. They were collected in the coldest part of the year. A table shows that for temperatures below 70° F. an increase of one degree in

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\* Dr. Andrew BALFOUR suggests that his mule trypanosome from Western Mongalla (see this *Bulletin* Vol. 1, p. 673) is *T. multiforme*.

the mean temperature caused a reduction of the pupation period of from three to five days. A list is given of the blood-sucking insects and ticks collected in the valley and on the plateau.

Two Appendices deal with an experiment to ascertain whether Tabanids transmit trypanosomes in nature, by WALLACE and LLOYD, and an attempt to transmit *T. rhodesiense* by means of *Ornithodoros moubata*, by A. F. WALLACE. The Tabanidae as they were caught were placed on a monkey; only a small proportion fed. The monkey did not become infected. All the flies that fed, viz., 128, were dissected and examined the same day. Flagellates confined to the mid and hind gut were found in seven.

For the tick transmission experiments the ticks had to be brought from the plateau, there being none in the valley; the natives say that it is too hot for them. Each group of ticks was fed on a *T. rhodesiense* monkey, and after an interval of about a month on a clean monkey. The following is a summary:—

125 ticks were fed on a heavily infected monkey.

After 1 month 87 were fed on a clean monkey.

„ 2	„ 51	„	„	„	„
„ 3	„ 8	„	„	„	„
„ 6	„ 23	„	„	„	„
„ 7	„ 10	„	„	„	„

The blood of the animals remained negative. 61 ticks were used in interrupted feeding experiments with uniformly negative results.

A. G. B.

YORKE (Warrington). i. **Sleeping Sickness and Big Game: A Proposed Experiment.**—*Brit. Med. Jl.* 1913. June 21. pp. 1315-1317.

ii. **The Relationship of the Big Game of Africa to the Spread of Sleeping Sickness.**—*Proc. Zool. Soc. of London.* 1913. June. pp. 321-337.

i. The author gives the gist of the findings of himself and KINGHORN in North Eastern Rhodesia. They proved that *G. morsitans* spreads the human trypanosomiasis of that country and found 1 in 500 of the flies caught in the Luangwa valley to be infective. They then examined a large number of wild animals, 698 in all, with a view to discovering the reservoir of the infection and found the trypanosomes of man or of stock in a considerable percentage of the antelope examined, 50 per cent. in the valley and 35 per cent. on the watershed. The corresponding figures for man were 16 and 3.3. Details are given in tables. The possible methods of dealing with the disease are then considered. It is concluded that the fly cannot be exterminated, nor the population removed from contact with it, and that the only possible course is to destroy the reservoir. Some of the objections that have been raised are considered. The author

favours an experiment of game destruction and says how in his opinion it should be carried out—

“A locality which is fairly well populated, and which contains plenty of tsetse fly and game should be chosen. An exact census of the population should be made, and the proportion suffering from trypanosomiasis determined. The same must be done in the case of the domestic animals, if such exist. An index of the percentage of infective tsetse fly must be ascertained. This is most important, as it gives a definite idea of the potential danger of the district. Finally, the game must be completely eradicated, and at the same time the percentage infected with the human and cattle trypanosomes determined, and when once the game has been driven out it must be kept back by vigorous action and not allowed to return. After an interval of some years the population, domestic stock, and tsetse fly must again be carefully examined. Then we should be in a position to decide definitely whether or not driving the wild fauna back from the sites of human habitations in fly areas would be advantageous on a general scale in tropical Africa. As such an experiment as this would take some years to accomplish, I consider that for the present the game laws should be removed in fly areas, and that Europeans and natives should be allowed to kill what they like, especially in the inhabited portions of these areas.”

He notes that it is proposed to depopulate the Sebungwe district, the focus of human trypanosomiasis south of the Zambesi. He thinks that this is wrong and that the game should be attacked with a view to stamping out the disease.

ii. The second paper traverses the same ground. An appendix contains the views of Fellows of the Zoological Society who took part in the discussion. [The opinions expressed had reference to the general question of game destruction rather than to the experiment advocated by Yorke.]

A. G. B.

**MACKENZIE (A.). Report on the Mafungabusi Fly Area. Southern Rhodesia.**—MS. Report to the British South Africa Company. Dated Jan. 29, 1913.

An expedition was undertaken with the object of defining the fly area in such parts of the Hartley and Mafungabusi districts as are traversed by the main footpaths from the Zambesi to Hartley. With respect to the type of country frequented by fly the author writes that he noticed the fly was almost entirely confined to the Mopani or else it was found in the bush in close proximity to a Mopani belt.

Throughout the journey all the natives met with at the various kraals were examined and blood smears taken from each. The slides, 500 in number, were all found to be negative.

In the fly area on the Mafungabusi side of the Umniati there was very little game, whilst on the Hartley side there was no sign of game except bushbuck in the bed of the river. The blood of numerous buck was examined for trypanosomes, but with negative results.

The only domestic animals seen were sheep, goats and dogs. The natives in the fly area informed the author that animals born in the neighbourhood survived, whereas imported beasts usually died. Numerous trypanosomes were found in the blood of one sheep.

W. Y.

## SLEEPING SICKNESS RESEARCH IN UGANDA.

**Reports of the Sleeping Sickness Commission of the Royal Society.**—  
No. 13.—ii. +142 pp. With 7 plates. 1913. London:  
H.M. Stationery Office. [Price 2s. 6d.]

This is a lengthy report containing eleven papers by Dr. H. L. DUKE and three by Miss ROBERTSON. Part of the work here recorded has already been published as separate papers in the *Proceedings of the Royal Society* and has received notice in this and the *Sleeping Sickness Bulletin*.

An account is here given of the six papers which have not been previously published.

**Notes on *T. gambiense* and *G. palpalis*.** By H. L. DUKE.  
pp. 13-21.

Experiments were undertaken to ascertain whether the nature of the blood ingested by the flies during the earlier days of an experiment had any effect on the development of *T. gambiense* in the fly. In this series of experiments *G. palpalis* were fed for 1 or 2 days on an infected monkey and subsequently, after starving for a day, on various clean animals—bushbuck, fowl, monkey, sheep, and calf. The flies were dissected and the number of positive ones determined. The results are summarised in the following table:—

Expt. No.	Percentage of positive flies with goat or buck blood.	Percentage of positive flies with cock or monkey blood.
	per cent.	per cent.
357 and 358 ...	11·5	2·6
567 „ 568 ...	3·5	0
581 „ 582 ...	20·6	3·6
617 „ 618 ...	3·7	0
127 „ 128 ...	0 (calf blood)	3·3

The percentage of positive flies is considerably greater in those experiments where the flies had been fed upon sheep or bushbuck blood. In spite of the contradictory result with the calf, the evidence of the first four experiments is sufficiently definite to afford a striking contrast to the results of KLEINE and FISCHER (*Sleeping Sickness Bulletin*, Vol. 3, p. 402) who obtained approximately 10 per cent. of infected flies when monkeys were used throughout the experiment as against 2·4 per cent. when ruminants were employed.

The obvious criticism of these results is that the difference is due to the flies having picked up flagellates other than *T. gambiense* from the sheep or bushbuck employed. Accordingly, control observations were made to prove that the sheep and bushbuck employed in the above experiments were free from sources of error. The conditions are doubtless very different at Tanganyika and Mpumu, both as regards the strain of trypanosome and the climate. This must account largely for the discrepancies

between the two sets of experiments. Moreover, as no reference is made by the German authors to the presence or absence of flagellates in the salivary glands, it is possible that some of their "*infizierte Fliegen*" showed no salivary infection and were hence incapable of infecting. Finally, the work of Miss ROBERTSON (*Sleeping Sickness Bulletin*, Vol. 4, p. 267) has demonstrated the occurrence of periods in the subject of trypanosomiasis when, quite irrespective of the mere presence of flagellates in the peripheral blood, the trypanosomes appear incapable of full development in the fly.

Observations were undertaken to ascertain the length of time laboratory bred *G. palpalis* survived starvation after feeds on various species of animals. There did not appear to be any marked difference in the various animals used. All except one fly were dead on the 18th day after the initial feeds. The remaining fly died on the 21st day.

An experiment was undertaken to show that *G. palpalis* infective for *T. gambiense* is capable of infecting monkeys on three consecutive days.

It was found that feeding a large number of negative *G. palpalis* on a monkey does not interfere with subsequent infection of the monkey with positive flies.

**The Sleeping Sickness Reservoir on the Islands of Lake Victoria, Nyanza.** By H. L. DUKE. pp. 54-57.

This paper deals with the problems relating to the continued infectivity of wild *G. palpalis* on the Northern Islands of Lake Victoria after the removal of the population.

A brief account of the distribution and general character of the islands is given. The only species of antelope found on these islands appears to be the sitatunga (*Tragelaphus spekei*). In several instances, since the removal of the island population, the antelope have migrated across the intervening channels to islands where they were formerly unknown. The distribution of *G. palpalis* is practically universal throughout the islands. Only the smallest rocks, almost bare of vegetation, are free from fly. The fly are found in large numbers on those islands where sitatunga abound, e.g., Damba and Bugalla of the Sese group, and Buziri near Buvuma.

An account is given of experiments to determine the infectivity of sitatunga and wild *G. palpalis* on certain of the islands. Wherever Glossina and buck occur together in numbers on an island, there the fly are found to be infective with *T. gambiense*. The degree of infectivity of the fly appears to be directly proportional to the frequency of exposure of the antelope to their bites. On islands where fly are very numerous and buck absent the former are apparently non-infective, even though hippos, reptiles and birds abound. All the facts point strongly to *Tragelaphus spekei* acting as a true reservoir for *T. gambiense* on these uninhabited islands; and the same doubtless is equally true as regards *T. vivax* and *T. uniforme*.



**Further Investigations on the Rôle of Antelope as a Reservoir of *T. gambiense*. By H. L. DUKE. pp. 58-66.**

Reference is made to a previous paper (*Sleeping Sickness Bulletin*, Vol. 4, p. 273) wherein it was shown (1) that antelope may remain capable of infecting *G. palpalis* with *T. gambiense* for at least twenty-two months after their original infection with this trypanosome; (2) that there is some evidence to show that an antelope which has ceased to be infective for *T. gambiense* acquires some degree of immunity against re-infection. Attempts were made to reinfect five antelope originally infected in April, 1910, which had shown no signs of infection for considerable periods. The attempts at reinfection were made in April, 1912. Of the five, two exhibited complete resistance, while the other three developed transitory infections. No protective bodies were demonstrated in the sera of these animals.

**A Trypanosome from British East Africa Showing Posterior-Nuclear Forms. By H. L. DUKE. (With a Note on its Developmental Stages in *G. palpalis*. By Muriel ROBERTSON.) pp. 67-89.**

This trypanosome was isolated from a donkey infected on the Wewe river. Details of its morphology are given. Briefly it may be described as a polymorphic parasite 12-38 $\mu$  long. The nucleus in a very considerable number of examples is situated posteriorly, close to the blepharoplast. The animal reactions of the parasite are given in tabular form. It proved virulent for monkeys, dogs, rats, rabbits, donkeys, goats, sheep and oxen: only one of six guineapigs became infected. The author points out that, except for its behaviour in guineapigs, the pathogenicity of the trypanosome corresponds roughly with that of *T. pecaui*. The lack of objective symptoms in sheep and goats appears to exclude *T. rhodesiense*. Experiments were undertaken to ascertain the action of human serum curatively and protectively on the infection in rats. The curative action was found to be weak and uncertain, but the protective action was much more marked, although complete prevention of infection was not observed.

*G. palpalis* fed on infected animals became infective and capable of transmitting the parasite to healthy animals. The cycle of infection in the fly was studied. After establishing their base in the "hind" and "fore gut" the flagellates advance towards the opening of the salivary gland ducts via the proventriculus. The earliest invasion of the salivary glands occurred on the 25th day when flagellates were seen in the proximal part of the gland and only rare colonies of attached individuals were observed further back; no forms were, however, to be found posterior to the proximal half of the organ. These flies were apparently unable to infect a monkey. The flies first became infective on the 26th day of the experiment. With the above exception every monkey which was fed upon by a fly showing flagellates in the salivary glands became infected.

The author continues by discussing the value of the manner in which trypanosomes infect the invertebrate host as a means of diagnosis. The results obtained by many workers have proved conclusively that the different species of mammalian trypanosomes do not all take up the same position in the alimentary tract of the intermediate host. Personal experience enabled the author to speak of 6 different species, viz., *T. gambiense*, *T. nanum*, *T. vivax*, *T. pecorum*, *T. uniforme*, and the subject of this paper. Before completing its development in the fly, each species takes up what may be termed its "anterior station," which in the case of the above is either the proboscis or salivary glands. In no case have developing flagellates been found in both situations simultaneously. On rare occasions a few trypanosomes may be found in the proboscis of flies showing salivary infection, but these almost certainly came from the gut or glands.

Of the above mentioned species *T. uniforme* and *T. vivax* are exclusively proboscis forms; *T. pecorum* and *T. nanum* show "infection totale" *sensu* ROUBAUD, and *T. gambiense* and the subject of this paper develop in the gut and salivary glands.

Comparison of the various species of trypanosomes dealt with by other observers, with these gives the following: (1) "Infection totale" of proboscis and gut—*T. congolense*, *T. pecorum*, *T. nanum* and *T. pecaui*; (2) Proboscis infection only—*T. vivax*, *T. uniforme* and *T. cazalboui*; (3) Infection of gut and salivary glands, none in the proboscis—*T. gambiense*, *T. rhodesiense* and this trypanosome.

Trypanosomes generally admitted to be very nearly allied, e.g., *T. pecorum* and *T. congolense*, behave similarly in the fly. Another striking point is that the type of development in the fly for a given species of trypanosome is apparently constant, irrespective of the species of tsetse concerned. These facts indicate that the distribution of the mammalian trypanosomes in the invertebrate host affords valuable evidence in considering the question of diagnosis.

The author points out that ROUBAUD and his colleagues found that *T. pecaui* developed in the alimentary canal and proboscis of *G. longipalpis* and *tachinoides*; this fact makes it impossible to suppose that *T. pecaui* and the subject of this paper are identical. For the present the author refrains from naming the parasite.

Miss Robertson in a note on the life cycle of the trypanosome in the alimentary tract of *G. palpalis* states that in the main the development of this parasite is very like that of *T. gambiense*; nevertheless there are certain differences which serve to distinguish the two forms. The earliest unmistakable development was noted on the 3rd to 4th day of the infection. The trypanosomes had increased somewhat in size and bulk and were extraordinarily uniform in type, the membrane was narrower than in the blood type, the free flagellum was short and the kinetonucleus lay somewhat anterior to the posterior extremity of the body. Division was in active progress and the parasites were present in large numbers in the "hind" and "midgut." The details of division are identical with those in *T. gambiense*. The forms

seen in these early days are always fully developed trypanosomes, and the suppressed crithidial forms described during the earliest divisions of *T. gambiense* (see this *Bulletin*, Vol. 1, p. 267) were never seen in this trypanosome. From the 7th day long slender trypanosomes begin to appear in the gut and about the 14th day they are seen in the proventriculus where they form the predominant and usually, but not always, the only type. At this stage in the cycle there is often a rather strikingly long period during which development does not progress. The salivary glands are finally invaded from the proventriculus by way of the hypopharynx. The slender free-swimming proventriculus forms were seen in the proximal part of the hypopharynx of a fly on the 27th day; the salivary glands were not, as yet, affected. Infection of the mammalian host does not occur until the salivary glands are invaded. The actual development of the trypanosome in the glands resembles that of *T. gambiense*. The gland infections show far higher numbers than in the cycle of *T. gambiense*, but the development in the glands occupied a considerably longer period than is the case with the sleeping sickness organism. The development of the parasite in the fly is illustrated by 36 figures.

**Some Attempts to Transmit *Trypanosoma gambiense* by Wild Stomoxys; with a Note on the Intestinal Fauna of these Flies.**  
By H. L. DUKE. pp. 89-93.

The flies used in these experiments were *Stomoxys nigra* and *S. calcitrans*. The flies were fed first for varying numbers of days on an infected monkey and then placed daily on clean monkeys until the termination of the experiment at the death of the last remaining fly. None of the monkeys became infected. In all 363 flies were dissected—those dying during the first four days of the experiment were discarded—but although no flagellates having crithidial or trypanosome structure were seen, two interesting organisms were discovered. One was a flagellate showing the structural characters of Bodo; the other a crescent shaped body, which had no motility and was often present in enormous numbers in the fly's gut.

**Notes on the Behaviour of a Polymorphic Trypanosome in the Blood-Stream of the Mammalian Host.** By Muriel ROBERTSON. pp. 111-119.

This paper deals with the trypanosome described above by DUKE. In general the cycle of the parasite in the blood of the vertebrate host was similar to that of *T. gambiense* (see this *Bulletin*, Vol. 1, p. 44). There is a definite general correlation between a high percentage of long forms and a high percentage of division; and there is an equally clear correlation between a high percentage of short forms and a low percentage of division. As in *T. gambiense* the survivors who weather the untoward conditions which carry off the majority of the parasites during the depressed period are drawn from among the short forms. Division is not found in specimens below 20-21 microns in length, and the earliest forms in which any sign of division can be

detected are definitely long forms. The long forms are those about to divide and they produce the short forms, in some cases directly, in others probably upon the intervention of another or several divisions.

W. Y.

#### DIFFERENTIATION OF TRYPANOSOMES.\*

ROUBAUD (E.). Evolution comparée des Trypanosomes pathogènes chez les Glossines.—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 435-441. With 3 figs.

This paper is an attempt to classify African trypanosomes from their development in Glossina. The behaviour of the parasites in Glossina may be divided into (1) Phenomena of culture, (2) Phenomena of evolution. The former are characterised by the persistence of the virus in the form of trypanosomes, whilst the latter are characterised by the cyclic transformation of the parasite into crithidial forms and then into the typical trypanosome forms. Both may be temporary (abortive) or permanent.

In the phenomena of temporary culture the trypanosomes pass into the posterior part of the midgut in a modified trypanosome form of large size. The culture hardly lasts more than 2 or 3 days. The author has only observed this kind of culture in *T. gambiense*, *T. dimorphon*, *T. congolense* and *T. pecaui* and never in the case of *T. brucei* (Zululand), *T. congolense*. [This appears to be an error as *T. congolense* is amongst the trypanosomes of the previous group.] *T. cazalbou* and *T. evansi*. Hence one is able to distinguish *T. brucei* and *T. cazalbou* from those of the former group.

This temporary intestinal culture may in certain cases become permanent, characterised by its persistence in the intestinal tract even during fasting. The culture extends from the posterior midgut to the anterior gut. The author has never seen infection of the rectum, cysts, or infection of the Malpighian tubes, but the anterior and midgut may be invaded in its entirety. It is this that the author calls 'infection totale.'

While temporary culture of 24 to 48 hours is constant, permanent culture is observed only in a limited number of flies which varies according to the species of Glossina, the specific character of the virus, and the locality.

Permanent infection of the intestine of Glossina may be considered as a phenomenon of culture, for it is not accompanied by important morphological modifications. The appearances are comparable to those seen in artificial cultures such as are described by THOMSON and SINTON (see this *Bulletin*, Vol. 1. p. 53). Moreover, these forms of intestinal cultures are not infective.

The phenomena of evolution are characterised by crithidial transformation in the salivary region. [The expression "milieu salivaire" presumably includes the proboscis as well as the

\* See also DUKE, p. 243.

salivary glands.] They may be temporary (abortive) or permanent. The phenomena of permanent salivary evolution are of three distinct types.—(1) That described as *direct fixation in the proboscis*. The parasites became fixed in the wall of the proboscis as short crithidial forms: these multiply and give rise to the typical salivary trypanosomes which lodge in the hypopharynx. (2) That described as *indirect fixation in the proboscis*. Here the proboscis infection is secondary to an "infection totale" of the intestine. *T. dimorphon* and *T. pecaui* belong to this class. (3) That known as *indirect fixation in the salivary glands*. This is peculiar to the two human trypanosomes *T. gambiense* and *T. rhodesiense*. The author refers to the work of Miss ROBERTSON on the mode of invasion of the salivary glands from the intestine via the proboscis (see this *Bulletin*, Vol. 1, p. 502).

In conclusion the author attempts to group the various pathogenic trypanosomes of Africa according to their method of development in the tsetse fly. He gives the following four fundamental groups (1) the *cazalboui-vivax* type, (2) the *dimorphon-pecorum* type, (3) the *pecaui* type, (4) the *gambiense-rhodesiense* type. Reference is made to the statement by BRUCE and his collaborators that *T. rhodesiense* is identical with *T. brucei*. The author states that so far as the monomorphic organism kept at the Pasteur Institute [Zululand strain] is concerned this hypothesis cannot hold, as the organism is incapable of even temporary intestinal culture in *Glossina*. [This supports the contention of STEPHENS and BLACKLOCK that *T. brucei* (PLIMMER and BRADFORD) is different from that of the Uganda ox, *T. ugandae* (see this *Bulletin*, Vol. 1, p. 662).\*]

W. Y.

MACKIE (F. P.). **The Investigation of Protozoal Diseases with Special Reference to the Differentiation of Trypanosomes.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras Nov. 18, 19 and 20, 1912.* pp. 271-280. 1913. Simla: Government Central Branch Press.

The object of this communication is to indicate lines on which the differentiation of trypanosomes may be undertaken.

Investigation should be carried out under the following headings: 1. Morphological characters; 2. Pathogenicity; 3. Natural transmitting agent; 4. The reservoir. Other less important methods of differentiation are: 5. Cultural characters; 6. Cross immunization; 7. Serum diagnosis; 8. Geographical distribution; and 8. Effect of treatment. The author proceeds to discuss the methods and to apply them to the differentiation of *T. pecorum*, *T. nanum*, *T. uniforme*, *T. vivax*, *T. brucei*, *T. evansi*, *T. ingens*, *T. elephantis*, and *T. gallinarum*.

In two tables are given the points by which these trypanosomes may be distinguished.

W. Y.

\* It might also be explained by the fact that this is a laboratory strain which has not been through the invertebrate host for many years and has in consequence lost the power even to survive in the fly. A. G. B.

## DIAGNOSIS.

HECKENROTH (F.) & BLANCHARD (M.). *Recherches sur les Propriétés du Sérum des Malades atteints de Trypanosomiase au Congo français.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 444-447.

The properties in question (protective, attaching, and trypanolytic) have already been worked at, chiefly with the serum of Europeans. The authors undertook the investigation of these properties in a large number of infected and non-infected negroes at Brazzaville. They used a virus brought from the Pasteur Institute, Paris, which killed mice regularly in a few days.

*Trypanolytic power.*—The trypanosomes and fresh serum were placed in contact at 37° C. for a period not exceeding one hour. Only total or sub-total trypanolysis was considered positive. The results in 172 cases are given. A certain number, 18 per cent., of non-infected gave a positive result, while treated cases gave 44 to 50 per cent. positive and untreated cases 78 to 93 per cent. positive. The greatest proportion of positive results was obtained in untreated cases in a bad physical condition. There is some evidence that the presence of trypanosomes in the blood favours the reaction.

*Attaching power.*—Inactivated serum, leucocytes and trypanosomes were placed in contact for 20 minutes at laboratory temperature. Only complete attachment was considered positive. The number of positive reactions was 20 per cent., the state of the patient not affecting the result.

*Agglutinating power.* This was observed only in 4.8 per cent. of 144 cases; 50 non-infected cases gave no agglutination.

*Protective power.*—MESNIL and RINGENBACH found that normal human serum had a protective power against the strain of *T. gambiense* which the authors were using. In order to avoid this action the sera were kept 10 to 20 days in the ice chest, by which time the normal action had disappeared.

One cubic millimetre of serum was mixed with one-fifth of a cubic millimetre of a dilution of trypanosomes which had ten to the field (Mg. = 350 Diam.). The whole was injected intraperitoneally into rats and mice.

The sera of 50 individuals, of whom 25 had trypanosomiasis, were tested. The normal sera gave no protection; 24 out of 25 infected sera protected. Non-treated cases in an advanced state gave the longest protection, treated cases were less active. Of the latter by only two was complete protection conferred, in six others delay in infection was caused.

The conclusions are:—Protective, attaching, and agglutinating powers are confined to the serum of sleeping sickness patients. Trypanolysis is more frequent with the serum of sleeping sickness patients than with normal individuals.

The different properties do not always co-exist in the same serum. Trypanolytic and protective powers are observed in the serum of patients with trypanosomes in the blood at the time of taking the blood. The serum of infected cases is almost always

protective, frequently trypanolytic, rarely attaching, and very exceptionally agglutinating.

The existence of these properties in the serum has no prognostic value, and only the protective power is of sufficient constancy to assist in the diagnosis of doubtful cases.

W. Y.

MESNIL (F.). *A Propos du Pouvoir protecteur des Sérums des Malades du Sommeil.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 447-451.

The sera of sixteen patients in thirteen of whom trypanosomes had been found, the remaining three being suspected cases of sleeping sickness, were examined as regards their prophylactic action in mice. A brief account of each of the sixteen cases is given.

The various sera were used in doses of from half to 1 cc. Seven of the thirteen sera obtained from the patients in whom trypanosomes had been found protected mice completely against infection. The serum of one of the doubtful cases obtained three days before using protected completely in doses of 1 cc. Fourteen days after removal it only retarded the infection in mice for four days. The sera of the other doubtful cases were without action.

W. Y.

#### CHEMOTHERAPY.

MORGENROTH & TUGENDREICH. *Zur Chemotherapie der Trypanosomeninfektion.* [On the Chemotherapy of Trypanosomiasis.]—*Berlin. Klin. Wochenschr.* 1913. Feb. 24. Vol. 50. No. 8. pp. 367-368.

The experiments described in this paper are a continuation of the chemotherapeutical experiments which have been carried on for some years, along definite lines, by MORGENROTH, HALBERSTÄDTER, R. LEVY, and KAUFMANN. The authors refer to their own and their colleagues' work on quinine derivatives, especially in relation to pneumococcal infection (MORGENROTH and R. LEVY), and trypanosomiasis in the mouse (MORGENROTH and HALBERSTÄDTER). The present experiments deal with the simultaneous application of several chemotherapeutically active substances which in combination give better results than they do individually. LAVERAN and MESNIL and many other experimenters have worked at this point and EHRLICH especially has frequently pointed out the importance of this fact. The good results obtained by TSUZUKI, who employed a combination of three separate remedies, are referred to (see *Sleeping Sickness Bulletin*, Vol. 3, p. 251).

MORGENROTH and ROSENTHAL observed that salicylic acid had a distinct action in trypanosomiasis. They determined to test systematically a large number of substances, and to raise as far as possible the somewhat feeble action of salicylic acid. This they have succeeded in doing by means of combination methods.

Ethylhydrocuprein was used in combination with sodium salicylate in the treatment of mice infected with nagana. The

quantity of the former drug used was so small as to be in itself of little value in clearing the trypanosomes, as shown in control mice. The latter drug proved in many experiments to have but slight effect on the parasites, even when used in the largest possible doses. The combination of the two caused a temporary clearance of the trypanosomes out of the blood. One mouse was cured (4 months' observation). Quinine itself whether in watery or oily solution proved far inferior to ethylhydrocuprein. A further series of experiments proved a combination of ethylhydrocuprein base, sodium salicylate, and a small quantity of salvarsan to have excellent effects. Salvarsan alone or in combination with sodium salicylate gave poor results, but salvarsan with ethylhydrocuprein gave definitely better effects. The three drugs together, however, given at a single injection cleared the trypanosomes out of the blood of seven mice heavily infected with nagana, and these animals remained free from trypanosomes (observation over 4 months). These excellent results should cause the adoption of the use of quinine derivatives, especially ethylhydrocuprein, and salicyl derivatives as an aid in the treatment of diseases in which at present salvarsan alone is used. The authors consider it justifiable as a result of these experiments to investigate the effect in disease in man of a combination of these three drugs, more especially as they have further found that ethylhydrocuprein frequently injected in small doses has proved capable of preventing relapses in trypanosome infected animals which had been insufficiently treated with salvarsan. This preventive effect occurred in the majority of infected animals, while the controls died after relapses. Should this method be applicable to man, the field of its utility is fairly wide—sleeping sickness, malaria, and possibly syphilis.

W. Y.

HARTOCH, ROTHERMUNDT, & SCHÜRMANN. *Beziehungen zwischen toxischen und chemotherapeutischen Wirkungen der Antimonpräparate im besonderen bei Dourine.* [The Relation between the Toxic and Chemotherapeutic Actions of Antimony Preparations, particularly in Dourine.]—*Centralbl. f. Bakt.* 1. Abt., Ref. 1913. June 14. Vol. 57. No. 14-22. pp. 174\*-179\*.

The general poisonous effects of antimony are very similar to those of arsenious acid. Irritation of the gastric mucous membrane with vomiting, fall of blood pressure, haemorrhage in the intestinal wall, derangement of the nervous apparatus of the heart and striped muscles, fatty degeneration of the large glands, and paralysis of central nervous origin are all symptoms produced by either arsenic or antimony.

In more recent times the work of CLOETTA and his school has added to our knowledge of the action of antimony. In contradistinction to arsenic it is not possible to produce a high degree of tolerance against antimony, and further the amount absorbed (absolutely as well as relatively) rather increases than diminishes with increasing duration of administration. This failure to produce tolerance through repeated administration of antimony—comparable to the failure to produce antimony fast strains of



spirochaetes and trypanosomes—is a point of considerable significance in the estimation of the therapeutic value of antimony. Systematic experiments with various preparations of antimony—trivalent and pentavalent—have demonstrated, according to BRUNNER, that the toxicity of individual preparations cannot be explained by an especially quick absorption. The poisonous, strongly active compounds and the slightly active show no essential difference in the quantity absorbed. All the highly toxic compounds contain the antimony in the trivalent form and conversely the non-toxic contain it in the pentavalent form. This relation between valency and toxicity is not peculiar to antimony, but applies in the case of other elements.

Trioxidin (antimony trioxide) is a striking exception in that although it contains the antimony in the trivalent form and is of great therapeutic activity it is nevertheless almost non-poisonous to mice, rats, guineapigs and rabbits (see this *Bulletin*, Vol. 2, p. 134). How far the larger animals and man will prove to be tolerant is, as yet, unknown. This question is of great importance since the larger animals are usually less tolerant to the various medicants, *e.g.*, atoxyl, used in the therapy of trypanosome infections. In view of the fact that 100 mgm. of trioxidin can be given per 10 gm. of body weight without producing symptoms of acute poisoning in mice, and that permanent sterilisation is caused by one hundredth part of this dose, it can be assumed that the question of tolerance is no serious objection to the employment of the remedy in large animals and man. Experiments are to be undertaken to decide the point.

Mice, rats, guineapigs and rabbits infected with dourine were treated with trioxidin or dimethylphenylpyrazolon-antimony ointment and the results obtained were as successful as in the case of infection with nagana and *T. gambiense* (*loc. cit.*). A couple of thorough inunctions of the above ointment caused the trypanosomes to disappear from the peripheral blood by the next day. As a rule there was no relapse.

No antimony was discovered in the organs of animals which had received an intramuscular injection of trioxidin, except in the muscles into which it had been injected. Here it was visible macroscopically.

The paper concludes with the remark that intramuscular injections of trioxidin and the inunction of metallic antimony or other insoluble compounds far surpass in efficacy all hitherto described remedial measures for trypanosomiasis.

W. Y.

LAMBALLE (F. W.). *Trypanosomiasis and Surra. A Preliminary Note upon the Effect of Pancreatic Enzymes upon the Trypanosome of Surra. With an Explanatory Note by J. BEARD, D.Sc.*—4 pp. 1913. June 9. Edinburgh: Otto Schulze & Co.

Lamballe treated three mules heavily infected with *T. evansi* with 3,750 units of Trypsin and 1,000 units of Amylopsin on each of two consecutive days. Three days later only parasites in various stages of disintegration were found in the blood films.

The next day no trypanosomes were seen. [As there is no record of further observations, no conclusion of value can be drawn from this note.]

W. Y.

#### TSETSE FLIES.

MACFIE (J. W. Scott). **The Distribution of Glossina in the Ilorin Province of Northern Nigeria.**—*Bull. Entomol. Research.* 1913. May. Vol. 4. Pt. 1. pp. 1-28. With 7 plates and 1 map.

During the year 1912 the author made a systematic study of the distribution of tsetse in the Ilorin province of Southern Nigeria. This district is approximately 6,300 square miles in extent and supports a population of some half million people. It lies in the south western portion of Northern Nigeria. An account of the physical features and climatic conditions is given.

In all 612 collections of tsetses were made from more than 500 different localities which are indicated on a map constructed by the author. The species noted were *G. palpalis*, *G. tachinoides*, *G. submorsitans* and *G. longipalpis*. The first two species occurred in all districts of the province, whilst *G. submorsitans* was limited to the eastern division.

*The western division.*—*G. palpalis* and *G. tachinoides* were the only species noted in this division. As a rule no distinction could be detected in the haunts of these insects, but on the Weru River *G. tachinoides* were found feeding on the author's horse in an open space some distance from the river where there were many *G. palpalis*.

In spite of the general occurrence of tsetse numerous herds of Fulani cattle are found throughout the greater portion of this division. It would be a matter of no great difficulty to render the roads almost everywhere comparatively free from tsetse fly owing to the relatively small numbers of these insects and the strict localisation of their haunts.

*The eastern division.*—All four species were found in this district, but *G. longipalpis* was only taken in a few widely separated localities; although tsetse haunt practically every river of the western district and nevertheless cattle are abundant, no Fulani herds are found in this division except in the extreme western portion. The author points out that there is one great difference between the two districts: whereas *G. morsitans* occurs all over the eastern district it is apparently absent from the western. He considers that it is this species which is inimical to live stock.

*The southern division.*—*G. palpalis* and *G. tachinoides* only were found. The former appears to be distributed all over the district, whilst the latter was caught at but a few places.

*The small race of G. palpalis found in Ilorin.*—This is by far the most common tsetse fly in Ilorin and although it differs markedly in external characters from any recognised species it

has been identified by both NEWSTEAD and AUSTEN as *G. palpalis*. It is a small fly with abdominal markings practically identical with those of *G. tachinoides*, with the exception that the buff colour of the paler areas is replaced by a very characteristic grey-blue tint. The male genitalia are identical with those of *G. palpalis*. Tsetse flies of this type were collected from every part of the province and were often found associated with typical *G. palpalis* and *G. tachinoides*. A coloured plate of the fly is given.

*Mites found on tsetse flies*.—In a few specimens of both *G. palpalis* and *G. tachinoides* minute red mites were found attached to the abdomen, thorax and legs. They were of two distinct types and probably belong to the genus *Trombidium*.

*Fulani cattle*.—Tables are given of the distribution of cattle in the Ilorin province. In addition to Fulani cattle a few dwarf cattle are met with in the southern and south-eastern districts. They are found in the neighbourhood of villages and towns in districts where Fulani cattle cannot exist, and have therefore been credited with a natural immunity to trypanosomiasis. The author found, however, that they succumb to infection with *T. brucei*, although in two cases recovery from infection with *T. vivax* was observed to occur.

*Trypanosomiasis*.—In Ilorin province human trypanosomiasis is either very rare or altogether absent. This may be due to the fact that the native population is relatively immune to the disease, or to the absence of the species of trypanosome which are pathogenic to man. The comparative scarcity of game, which has been proved by KINGHORN and YORKE to be the reservoir of sleeping sickness in Rhodesia, should also be remembered in considering this subject. Trypanosomiasis of domestic animals is exceedingly common. Trypanosomes presenting the morphological characters of *T. brucei* (probably *T. ugandae* of STEPHENS and BLACKLOCK), *T. vivax*, *T. nanum* or *T. pecorum*, and *T. theileri* were found. The paper closes with some suggestions as to the manner in which trypanosomiasis might be dealt with in this province.

A synopsis in tabular form is given of localities in which the various tsetse flies were taken.

W. Y.

ROUBAUD (E.). Supplément à la Répartition et à la Variation Géographique des Glossines.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 347-350.

The author has revised the collections of Glossina at the Pasteur Institute and Paris Museum.

The great majority of tsetse from the French Congo are *G. tabaniformis* Westwood. True *fusca* is rare in the Congo.

In West Africa *G. fusca* is widely distributed on the Ivory coast and *G. nigrofusca* and *G. medicorum* are also found in this district. The last species also occurs in Dahomey, where it is met in the same places as *G. palpalis* and *G. tachinoides*.

Among the *morsitans* group are two examples of *G. austeni* which were taken in 1906 in Mozambique. An examination was

made of the male armatures of *G. morsitans* from West Africa, Chad, Nyasaland, and from the valley of the Limpopo. *G. morsitans* from West Africa and those from Chad are a different variety from those of East Africa. The name *submorsitans* proposed by NEWSTEAD (*Sleeping Sickness Bulletin*, Vol. 3, p. 80) may serve to distinguish the western variety from the other. The distinction between these two geographical sub-species is analogous to that which exists between the eastern *G. pallidipes* and the western *G. longipalpis*.

*G. palpalis* also vary in different localities. Those from West Africa are larger (9-10 mm.) and of dark colour and the superior claspers are highly developed, while those from East Africa are smaller and of a lighter colour. The species described by Newstead as *G. fuscipes* ought to be regarded as a geographical variety of *palpalis*. This form appears frequently in Uganda and it is also represented in the collection at the museum, the specimens having come from the Belgian Congo.

W. Y.

NEWSTEAD (R.). **A New Tsetse Fly from the Congo Free State; and the Occurrence of *Glossina austeni* in German East Africa.**—*Ann. Trop. Med. & Parasit.* 1913. June 10. Vol. 7. No. 2. pp. 331-334.

A description is given of a new species of tsetse fly from the Congo, *Glossina severini*, of which two examples were available.

The author remarks that the species can be easily separated from *G. fusca* by the much darker colour of the pleura and the hind coxae; in these characters it resembles *G. fuscipleuris*, but it has relatively longer and stouter palpi and is also distinguished from the latter by its darker and more uniformly coloured hind tarsi.

The male genital armature of *G. severini* resembles closely that of *G. medicorum*, but it can be separated from the latter by its rudimentary harpes, the greater length of the marginal hairs of the editum, and by the greater length of the lateral branches of the hairs of the arista.

There are figures of the male armature of *G. severini* and of the terminal portions of the hairs of the arista of this species and of *G. medicorum*.

The author records that he has obtained twelve examples (2 ♂ and 10 ♀) of *G. austeni* (this *Bulletin*, Vol. 1, p. 523) from the Tanga district of German East Africa.

W. Y.

SHIRCORE (J. O.). **On Two Varieties of *Glossina morsitans* from Nyasaland.**—*Bull. Entomol. Research.* 1913. May. Vol. 4. Pt. 1. p. 89.

*Glossina morsitans*, Westw., var. *pallida*. nov.

The colour markings of this tsetse—a male taken in the Dowa district of Nyasaland—are described in detail. The author remarks that the fly was picked out at a glance from more than

a hundred *G. morsitans* and is distinctly and remarkably paler throughout. The last two joints of the hind tarsi are faintly dark, but not nearly so dark as in *G. morsitans*.

In a footnote it is stated that there is in the British Museum a single male (also from the Dowa district) which agrees entirely with this specimen. The genitalia are indistinguishable from those of typical *G. morsitans*.

*Glossina morsitans*, Westw., var. *paradoxa*. nov.

This specimen—a male—which was caught in the same district resembles superficially *G. morsitans*, but the hind tarsi are entirely dark as in the *palpalis* group. The superior claspers of the male genitalia resemble those of *G. submorsitans*, but are more deeply pigmented throughout, especially along the lateral and posterior borders.

W. Y.

#### TRYPANOSOMA CRUZI INFECTION.

CHAGAS (Carlos). **Les Formes Nerveuses d'une Nouvelle Trypanosomie** (*Trypanosoma cruzi* inoculé par *Triatoma magista*) (**Maladie de Chagas**). — *Nouvelle Iconographie de la Salpêtrière*. 1913. Jan.-Feb. Vol. 26. No. 1. pp. 1-9. With 7 plates.

This paper is a résumé of the author's observations on the nervous manifestations of the disease called after him. Most of the observations recorded have already been dealt with in previous papers (*Sleeping Sickness Bulletin*, Vol. 4, p. 341, and this *Bulletin*, Vol. 2, p. 138). Writing on the frequency of the nervous forms of the infection the author says "We have numerous observations, which entitle us to state that this malady is the one which causes perhaps the greatest number of organic affections of the nervous system in human pathology."

A case of cerebral spastic paraplegia is described in detail in which there were also symptoms of cerebellar origin.

The paper is illustrated by numerous photographs.

W. Y.

BRUMPT (E.) & GONZALEZ-LUGO. **Présentation d'un Réduvide du Vénézuëla, le *Rhodnius prolixus* chez lequel évolue *Trypanosoma cruzi*.**—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 382-383.

*Trypanosoma cruzi* was found in the faeces of *Rhodnius prolixus* more than two months after they had been fed on an infected monkey.

The authors write that if *Rhodnius prolixus* maintains its infection indefinitely as does *Conorhinus megistus*, and not during some months only as *Cimex lectularius* and *Cimex rotundatus*, these insects are still more to be feared than the *Conorhinus*. For whereas the latter does not defecate as a rule for some seconds or even minutes after feeding, the *Rhodnius*—both larvae

and nymphs—defecates immediately after having withdrawn its proboscis, and hence the individual bitten has much more chance of being infected.

*Rhodnius prolixus* swarms in certain portions of Venezuela, Columbia, and the adjoining countries.

W. Y.

UNCLASSIFIED.

HECKENROTH (F.) & BLANCHARD (M.). *Transmission du Trypanosoma gambiense par des Moustiques (Mansonia uniformis)*.—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 442-443.

Experiments were conducted at Brazzaville, French Congo, to ascertain whether the mosquito (*Mansonia uniformis*) could transmit *T. gambiense*. [See *Sleeping Sickness Bulletin*, Vol 1, p. 70.]

A mosquito proof cage was divided into two compartments by a partition which extended about two thirds the height of the cage. Into each of these a guinea-pig was placed—one infected and the other normal. Mosquitoes were then introduced into the cage. After 48 hours the healthy animal was withdrawn. It subsequently was found to be infected.

In a second experiment 26 hungry *Mansonia* were allowed to feed on a heavily infected guineapig. After 24 hours the infected animal was withdrawn and 24 hours later a healthy guineapig was introduced into the cage. After a further interval of 23 hours this was also withdrawn. It subsequently showed trypanosomes in the peripheral blood.

This experiment shows *Mansonia* can retain the power of infecting for at least 24 hours.

W. Y.

TODD (John L.). *Concerning the Sex and Age of Africans suffering from Trypanosomiasis*.—*Ann. Trop. Med. & Parasit.* 1913. June 10. Vol. 7. No. 2. pp. 309-319.

Of the 79 cases of trypanosomiasis seen by the author in the Gambia (*Sleeping Sickness Bulletin*, Vol. 3, p. 336), 76 per cent. (39·3 per cent. males and 36·7 females) were adults and 24 per cent. children (16·4 per cent. boys and 7·6 per cent. girls); none were aged.

Slight degree of glandular enlargement is common in children of both sexes. Much enlarged '+' glands occur most often in young adults and trypanosomes were found in every person with much enlarged glands who was examined for them.

Almost 90 per cent. (60 per cent. males and 30 per cent. females) of the 416 cases of trypanosomiasis seen by DUTTON and the author in the Congo were young adults. With the exception that the percentage of infected young people is greater, the incidence of trypanosomiasis in the Gambia differs slightly from that in the Congo.

The rareness of the disease amongst old people is striking. Even although a large proportion of adults infected with trypanosomiasis and untreated die within 3 or 4 years and although the aged are not exposed to infection by their occupation, it is astonishing that the percentage of infected old persons should be so small; it seems impossible that even so small a number of persons could live during their years among populations so heavily infected without themselves contracting the disease. The only explanation which suggests itself is that some natives may be immune to trypanosomiasis. If an immunity does exist it is probably not an absolute 'sterilizing' immunity, but a relative one in which the host is tolerant of the infecting parasite.

The following are the conclusions:—

1. The proportion of elderly individuals among the natives of the Congo and Gambia is lower than it is among Europeans.
2. By far the majority of cases of trypanosomiasis are persons of middle age; almost none of them are elderly persons.
3. The percentage of individuals with a considerable degree of glandular enlargement—which is coincident with trypanosomiasis—is very much greater in adults, and in children, than in elderly persons.
4. It is possible that the low incidence of trypanosomiasis among elderly persons may be due, in part at least, to an immunity acquired by them.

W. Y.

**McCOWEN.** A Note on Sleeping Sickness in Principe Island and Angola, West Coast of Africa.—*Proc. Roy. Soc. Med. (Sect. of Epidemiology & State Med.)* 1913. May. Vol. 6. No. 7. pp. 191-194.

This paper gives a brief account of Sleeping Sickness in Principe and Angola.

In a footnote BASSETT-SMITH states that the great majority of the tsetse collected by McCowen were *Glossina palpalis*. There were two specimens of the variety *G. palpalis wellmani* and one example of *G. medicorum* which is new to the district.

W. Y.

**HIRSCH (Rahel).** Trypanosomen-Wärmestich-Anaphylatoxinfieber beim Kaninchen. [Trypanosome-Isolation-Anaphylatoxin Fever in Rabbits.] — *Zeitschr. f. Exper. Patholog. u. Therapie.* 1913. Apr. 24. Vol. 13. No. 1. pp. 132-142.

The author gives an account of experiments which she undertook in connection with heat production, oxygen absorption, and carbonic acid excretion in experimental animals. Rabbits were used and the condition of fever produced by infection with trypanosomes, insolation, and anaphylatoxin. Heat production was tested by the direct method, and the absorption of oxygen and excretion of carbonic acid at the same time ascertained. The calorimeter was put at 15.8° C., as in all previous experiments. Rabbits give a very regular curve of heat production, so that animals of the same body weight on an equal diet give practically identical curves.

The results of the experiments are clearly set out in 28 tables and the conclusions are as follows:—

1. Fever due to infection such as is caused by trypanosomes evokes in Rabbits an increased heat production.

2. Insolation also brings about an increase in heat production which is slighter than that observed in Trypanosome fever.

3. In Anaphyatoxin fever even with high temperature the heat production in Rabbits may fall far below the normal.

W. Y.

**HIRSCH (Rahel).** Fieber und Chininwirkung im Fieber. [Fever and the Action of Quinine in Fever.]—*Zeitschr. f. Experiment. Patholog. u. Therapie.* 1913. Apr. 24. Vol. 13. No. 1. pp. 84-131.

This is a highly technical paper dealing with metabolism and heat production in fever, and the effects of quinine administered during the continuance of the fever upon them. A summary of previous work done on this and kindred subjects is given. The author's own observations were made upon two dogs. These were put on a fixed diet and studied in the state of health. They were then inoculated with pathogenic trypanosomes, Nagana in one case and *T. brucei* (PROWAZEK'S) in the other, and were studied during the febrile period which lasted with short intermissions until their death.

Details of diet, technique, and apparatus used are fully given and the results are summarized clearly in Tables. [Those interested in the subject of metabolism should read this paper in the original.]

W. Y.

**BROWN (Alexander).** Native Treatment of Sleeping Sickness—A Trial.—*Jl. Trop. Med. & Hyg.* 1913. June 2. Vol. 16. No. 11. p. 167.

The remedy, a decoction of the bark of a tree called "Musolkwe," was found to be of no value.

W. Y.

**KOHL-YAKIMOFF (Nina), YAKIMOFF (W. L.), & SCHOKHOR (N. J.).** Le Trypanosome des Bovidés (*Tr. theileri* ou du Type voisin) au Turkestan.—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. p. 434.

Cattle in Turkestan are heavily infected with *Theileria mutans*, but the authors were unable to find in the peripheral blood *Trypanosoma theileri*, although they examined 154 animals in the municipal slaughterhouses of Taschkent. No person, they state, has seen these flagellates in this district. By means of inoculating tubes of ordinary broth with blood from the jugular vein, cultures were obtained from 8 animals out of 9. The amount of blood used was 4-5 cubic millimetres per tube.

W. Y.



KOHL-YAKIMOFF (Nina), YAKIMOFF (W. L.), & BEKENSKY (P. W.).  
**Le Trypanosome des Bovidés (*Tr. theileri* ou du Type voisin)  
en Russie d'Europe.**—*Bull. Soc. Path. Exot.* 1913. June.  
Vol. 6. No. 6. pp. 433-434.

The authors worked at St. Petersburg with cattle (from the provinces of Witebsk and Pskow), in which parasites were not visible in the peripheral blood. They used the method of Ed. and Et. SERGENT. Cultures were obtained from 5 of 14 animals examined. The culture was easily carried on from broth to the NNN medium for several generations. Finally, they inoculated the broth cultures into a calf and this became infected.

W. Y.

LAVERAN (A.) & FRANCHINI (G.). *Trypanosoma talpae* chez *Palaeopsylla gracilis*.—*Compt. Rend. Soc. Biol.* 1913. June 20. Vol. 74. No. 22. pp. 1254-1256 With 11 text-figs.

The authors examined the fleas, *Palaeopsylla gracilis*, found on moles, and in 10 out of 36 found flagellates which they consider cultural forms of *Trypanosoma talpae* in the digestive tract of the fleas. Parasitised fleas were taken, 8 out of 10 times, from moles in which *T. talpae* was found. The stages of the parasite encountered are described as (1) small, free, oval elements; (2) fusiform elements, sometimes with signs of bipartition in their nuclei and often forming rosettes containing a variable number of organisms; (3) *Leptomonas*-like forms; (4) typical small trypanosomes with a somewhat straight undulating membrane. The various phases recall those of *T. lewisi* in the rat flea. The authors recognise the existence of natural flagellates of fleas but consider that the presence of definite small trypanosomes excludes their organism from the category of natural flagellates. The authors illustrate each form of organism described.

A. Porter.

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## PROTOZOOLOGY.

RODHAIN (J.), PONS (C.), VANDENBRANDEN (F.), & BEQUAERT (J.).  
**Notes sur quelques Hématozoaires du Congo Belge.**—*Arch. f. Protistenkunde*. 1913. Apr. 19. Vol. 29. No. 2. pp. 259-278. With 1 plate and 5 text-figs.

The authors describe briefly various Haematozoa found by them on their journey from Leopoldville to Katanga, Belgian Congo. The parasites include trypanosomes from two genera of mammals and five of birds; haemogregarines from three genera of snakes, from one *Varanus niloticus* and from one *Bufo* sp., while 3 genera of mammals were also parasitised; a *Leucocytoegregarina* from the jackal, *Canis adustus*; a new *Plasmodium* from the insectivore *Petrodromus tetradactylus*, both gametocytes and schizonts being described; *Halteridium* and *Haemoproteus* from 22 different genera of birds; and Leucocytozoa from 14 avian genera. Full lists of the respective hosts are given, together with brief details regarding the parasites.

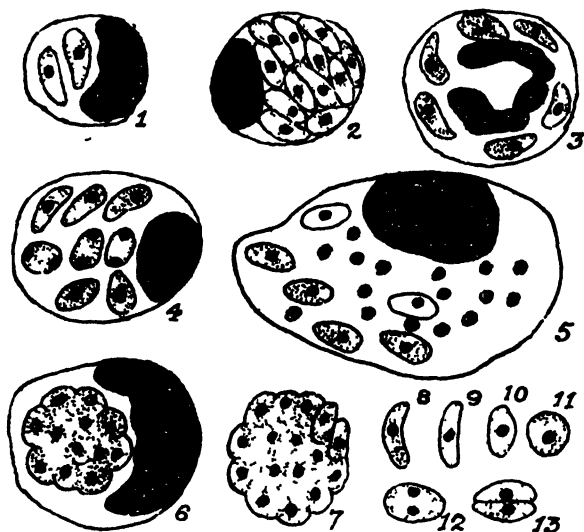
A new trypanosome from the Macroscelid insectivore *Petrodromus tetradactylus* is designated *T. brodeni*, but the avian trypanosomes are grouped according to their hosts only. The same applies to the haemogregarines, halteridia and Haemoproteus. Of the Leucocytozoa, that from the great heron, *Ardea goliath*, is termed *Leucocytozoon ardeae*, and that from the fowl, *L. schoutedeni*. No new name is given to the others described, the authors remarking [and most justly] that until the mode of transmission is known [as well as their full developmental cycle] new names must be considered as provisional only. The paper is well illustrated.

H. B. Fantham.

- i. LAVERAN (A.) & MARULLAZ (M.). **Contribution à l'Etude morphologique du *Toxoplasma gondii* et du *T. cuniculi*.**—*Compt. Rend. Acad. Sci.* 1913. Séance du 28 Apr. Vol. 156. No. 17. pp. 1298-1302.
- ii. LAVERAN (A.). **Présentation d'un Chien infecté de Toxoplasmose.**—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. p. 294.
- iii. LAVERAN (A.) & MARULLAZ (M.). **Recherches expérimentales sur le *Toxoplasma gondii*.**—*Ibid.* June. No. 6. pp. 460-468.

i. The authors consider the morphology of these species separately. (1) *Toxoplasma gondii* was studied in peritoneal exudate and smears of organs. The uninucleate parasite is usually oval but its shape and size may vary. The length is  $2.5\mu$  to  $6\mu$ , and the breadth is  $2\mu$  to  $3\mu$ . Multiplication is by binary fission.

The parasites occur in mononuclear and polymorphonuclear leucocytes, the endothelial cells of the peritoneum and splenic capillaries, and in hepatic cells [vide diagrame]. The number of parasites in a host cell may be few or as many as 30. Pseudocysts are aggregations of parasites. 13 text figures are given.



*Toxoplasma gondii*, endocellular or free in the peritoneal exudate of infected mice

1. Mononuclear containing two toxoplasms. 2 Mononuclear filled with toxoplasms. 3 Polynuclear containing toxoplasms. 4. Endothelial cell containing toxoplasms, three of which are dividing. 5. Endothelial cell containing toxoplasms, some with clear outline and others of which the nuclei only are seen. 6 Endothelial cell with agglomerated toxoplasms. 7. Agglomeration of toxoplasms, the contours of two are seen. 8-11. Free toxoplasms. 12-13. Division stages. (Magnification about 1,600 diameters.)

(2) *Toxoplasma cuniculi*.—The dimensions are much the same. The authors cannot agree with SPLENDORE who thought that some specimens possessed a blepharoplast and flagellum, nor do they agree with his and CARINI's account of multiplication by schizogony.

Laveran and Marullaz consider that there is insufficient evidence from the morphological side to justify the separation of the two species.

ii. Laveran exhibited a young dog successfully inoculated intravenously with diluted peritoneal exudate from a mouse infected with *Toxoplasma gondii*. For the first month the dog lost weight, then gained again. The cornea gradually became opaque but total opacity did not occur; purulent mucous discharge came from the nostrils. Very few parasites have been seen, though one leucocyte contained nine. Laveran points out that it is of interest that canine toxoplasmosis causes temporary corneal opacity as in some trypanosomiases.

H. B. F.

iii. This paper contains a full account of the various animal reactions of *Toxoplasma gondii*, as investigated by the authors. Details of each case are given. The authors have successfully inoculated the mouse, *Mus sylvaticus*, *Microtus arvalis*, the rabbit, guinea-pig, mole, hedgehog, shrewmouse, dog, pigeon, and *Padda oryzivora* with *Toxoplasma gondii*. They have failed to reproduce the disease in the rat, dormouse, fowl, several exotic passerines, frogs and lizards. Previously the authors have considered that *T. gondii* and *T. cuniculi* are identical from the similar morphology of the two organisms (see this *Bulletin*, Vol. 2, p. 52). They are strengthened in this opinion by the similarity of the animal reactions, each virus showing the same peculiarities, *e.g.*, inoculability to animals belonging to different classes (mammals and birds), and the susceptibility and refractoriness respectively of such nearly allied animals as the mouse and the rat.

A. Porter.\*

SPLENDORE (A.). Des Formes flagellées et des Gamètes dans le *Toxoplasma cuniculi*.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 318-323. With 1 plate.

Splendore refers to his previous notes on *Toxoplasma cuniculi* in which he expressed a belief that the organism had a flagellate stage. He has now seen in the liver smears of an infected pigeon a parasite possessing what he describes as a filament, which passes along the convex edge of the parasite, makes a slight curve, traverses the attenuated extremity of the body and is free for about 14  $\mu$ . Its appearance suggested a malarial microgamete, but it is not of this nature, since Splendore has seen what he considers as undoubted gametes in smears of the pectoral muscles of a pigeon. The infected breast muscles showed greyish white spots and various stages of *Toxoplasma* were infiltrated therein. The parasites were found in dry smears stained with Giemsa. They reach 10  $\mu$  in diameter and are roundish, while ordinary forms are 7 to 8  $\mu$  by 2 to 3  $\mu$ . The cytoplasm is attenuated and contains rodlets of chromatin explained as the result of nuclear reduction. Some showed two, others four nuclei, the results of division of the primitive nucleus. Two chromatoid filaments arise near the nuclei and sometimes reach 3 to 4  $\mu$  beyond the body. By analogy with the malarial parasites, Splendore considers that the flagellate forms represent microgametes. He does not at present definitely state which are the female forms. In conclusion he states that the above suppositions will be verified when the sexual forms are found in the invertebrate host, which he has incriminated as a *Stomoxys*.

H. B. F.

\*Dr. A. PORTER is in charge of the Protozoological Section during Dr. FANTHAM's absence in Khartoum.

MESNIL (F.) & SARRAILHÉ (A.). *Toxoplasmose expérimentale de la Souris; Passage par les Muqueuses; Conservation du Virus dans le Cadavre.*—*Compt. Rend. Soc. Biol.* 1913. June 27. Vol. 74. No. 23. pp. 1325-1327.

The authors worked with *Toxoplasma gondii*, using a strain from NICOLLE and Mme. CONOR. The first experiments were to determine whether toxoplasms could traverse the uninjured mucosa of mice. In a preliminary experiment a drop of ascitic fluid was placed in the vagina and on the conjunctiva respectively of two mice, both of which became infected. The table shows the results of the subsequent experiments.

Mucous Membrane.	Results.		Average Duration of Infection.
	Positive.	Negative.	
Vagina ... ..	4	0	10 days.
Prepuce ... ..	0	2	—
Conjunctiva ... ..	1	0	13 and a half days.
Vagina and conjunctiva	2	0	10 days.
Mouth ... ..	2	1	14 and a half days.

Every precaution was taken to avoid lesions of the mucosa. The toxoplasms, though able to penetrate the mucosa, were unable to pass through the uninjured skin. Generalised infection followed infection per mucosa and the incubation period was extended. The quantity of virus inoculated made but little difference to the period necessary for a fatal effect. All the organs of the mice showed some toxoplasms but abundance of parasites occurred in the thymus and hypertrophied lymphatic glands.

Local inflammation accompanied vaginal infection. Sero-purulent, viscous liquid containing leucocytes and free or intracellular toxoplasms exuded some days after the introduction of the virus. The contained parasites had not lost their virulence.

Ascitic fluid retained its virulence till about eighteen hours after the death of the host; on one occasion it persisted for 30 hours.

A. P.

MARULLAZ (M.). *Au Sujet d'un Toxoplasma des Oiseaux.*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 323-326.

The author has studied the small parasites that coexist in *Padda oryzivora* infected with *Haemamoeba danilewskyi* and were previously noted by LAVERAN, ADIE, and de BEAUREPAIRE-ARAGAO. He considers the parasite to be a new species of *Toxoplasma*. He states that its occurrence in birds quite free from *H. danilewskyi* and other haemotozoa, its endoleucocytic habit, its method of multiplication and absence of pigment, makes it impossible to consider it an avian *Haemamoeba*, while its multiplication differentiates it from the haemogregarines.

On the other hand, the form of the organism, its dimensions, staining reactions, nuclear structure, longitudinal division, and presence in the mononuclear leucocytes of the viscera and the

blood, permit its inclusion in the genus *Toxoplasma*. Attempts to inoculate the parasite to the pigeon and mouse failed. This, together with absence of crescent-shaped parasites, serves to differentiate it from *T. gondii* and *T. cuniculi*. The author therefore names it *T. avium*. The paper is illustrated by 9 text-figures of *T. avium*.

H. B. F.

SANGIORGI (Giuseppe). *Un Nuovo Protozoo Parassita del Mus musculus.*—*Pathologica*. 1913. June 1. Vol. 5. No. 110. pp. 323-325.

The author briefly discusses the genus *Toxoplasma*, and the species known at present, namely *T. cuniculi*, *T. gondii*, *T. canis* and *T. talpae*.

Sangiorgi found a toxoplasma in the mouse, *Mus musculus*, especially in the spleen and liver; it was absent from the blood. It was usually extracellular, rarely in mononuclear elements, measuring about  $3.5\mu$  in diameter, and rather smaller than allied species. Its shape was round, oval, pyriform, reniform or crescentic. There was a single nucleus in non-dividing forms. The author names the parasite *Toxoplasma musculi*. There are 12 text-figures, some showing longitudinal bipartition.

H. B. F.

i. PHISALIX (M.) & LAVERAN (A.). *Sur une Hémogrégarine nouvelle, de Lachesis alternatus.*—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 330-333. With 12 text-figs.

ii. PHISALIX. *Sur une Hémogrégarine du Python molure et ses Formes de Multiplication endogène.*—*Compt. Rend. Soc. Biol.* 1913. May 23. Vol. 74. No. 18. pp. 1052-1054.

iii. *Sur une Hémogrégarine de la Vipère Fer de Lance et ses Formes de Multiplication endogène.*—*Ibid.* June 20. No. 22. pp. 1286-1288. With 11 text-figs.

i. A new haemogregarine designated *Haemogregarina roulei* occurring in the blood of the viper, *Lachesis alternatus*, is described by the authors. Three forms of parasites occur within the erythrocytes:

1. Small forms, generally two or three at least in each infected cell. 2. Medium sized forms. 3. Large forms representing the adult parasites. Free forms are represented by: (a) very small vermicules, and (b) large adult forms.

No multiplicative stages have been found in the circulating blood, and the death of the host is awaited before search of the internal organs can be made.

It is interesting to note that as many as five parasites may be enclosed within one erythrocyte.

The text figures illustrate various aspects of *H. roulei* in the blood of the host.

ii. The subject of research was *Haemogregarina pococki* (Sambon and Seligmann) in *Python molurus*. The parasite is closely allied to the haemogregarine described by LAVERAN and PETTIT in *Python sebai*.

Endoglobular, free and multiplicative forms are figured, and their dimensions given. Multiplication cysts are only found in the hepatic capillaries. The cysts are ovoid or spherical, and may contain four merozoites. Haemogregarines also occur in the large pigmented elements of the hepatic capillaries.

The paper is illustrated by 15 text figures, showing the various forms described.

H. B. F.

iii. A haemogregarine was observed by Phisalix in the blood of one of two specimens of the viper, *Lachesis lanceolatus* (= *Trigonocephalus lanceolatus*, Oppel, *Bothrops lanceolatus*), received at the serpentarium of Butantan, Brazil. Larvae of worms in the perivisceral conjunctive tissue, large colourless nematodes from the oesophagus and stomach and minute nematodes from the intestine yielded no stages of the haemogregarine.

Endoglobular forms usually occurred singly in the red cells. If two or three parasites were present, the host cell hypertrophied; if one parasite only were present no change of size or shape usually occurred. The parasites commonly were recurved within the cell. Free forms were vermicular and were sometimes encapsuled, sometimes naked. Multiplication by cysts of the parasite is described for the first time, a few cysts having been seen in capillaries of the liver when sectioned. The encysting organism increases in size, its contents become very granular, and two to four nuclei may be present within the thick wall. Two macromerozoites have been found in ripe cysts. It is very rare indeed to find such cysts, which at first were undetected and hence are not depicted.

A. P.

THIROUX (A.). Les Formes de Reproduction par Schizogonie et Sporogonie d'*Haemogregarina pettiti* (Thioux, 1910) chez *Crocodilus niloticus*.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 327-330. With 10 text-figs.

The author refers to his previous paper (1910) on *Haemogregarina pettiti* occurring in the crocodile of St. Louis, Senegal. He now has found in the liver of the host that two forms of cysts occur. The first form is thickwalled and is considered by the author to be an oöcyst containing four ovoid spores, each of which may form two sporozoites. The second form consists of cysts containing eight merozoites distributed irregularly within, such cysts forming the schizogonic cycle.

The author thinks that he has thus demonstrated the occurrence of schizogony and sporogony within the vertebrate host, without the aid of an invertebrate transmitter.

The text figures illustrate the two kinds of cysts described.

H. B. F.

MOLDOVAN (J.). *Sur le Développement du Leucocytozoon ziemanni* (Laveran).—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 428-429.

Moldovan examined the internal organs of two owls, *Athena noctua*, heavily infected with *Halteridium noctuae* and *Leucocytozoon ziemanni*. In stained specimens he found in addition to typical gametes, forms which he considers to be schizonts of *L. ziemanni*. They occur in mononuclear cells (lymphocytes or erythrocytes), and in lung and brain smears, all stages from uninucleate forms to schizonts containing 30 nuclei were seen. Merozoite formation, however, was not observed. Some young parasites present were considered to be gametes because of their staining reactions. Trypanosomes described as male and female occurred in the same films. The schizonts were assigned to the genus *Leucocytozoon* rather than to that of *Halteridium* (which was also present in the hosts) on the grounds of the form and structure of the host cells and of the parasite before division, and their resemblance to forms described by other workers for the Leucocytozoa of fowls.

A. P.

FRANCHINI (G.). *Nouvelle Contribution à l'Etude de Haemocystozoon brasiliense*.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 333-336. With 5 text-figs.

Franchini has confirmed his previous opinion (see this *Bulletin*, Vol. 2, p. 46) that *Haemocystozoon brasiliense*, parasitic in the blood of man, belongs to the Flagellata. He has found non-flagellate and encysted stages of the parasite in smears of blood obtained from liver puncture. He describes and sketches:

1. Oval or lanceolate forms, measuring  $3\mu$  to  $6\mu$  long by  $1\mu$  to  $2.5\mu$  broad. They possess neither blepharoplast nor flagella.



*H. brasiliense*. The figure on the right is from a coloured plate.

2. Oval or more often lanceolate forms, sometimes seen dividing, some reaching  $16\mu$  in length and  $3\mu$  in width, most being  $12\mu$  long.

3. Flagellate forms with flagella up to  $20\mu$  long, arising near the blepharoplast.

4. Non-flagellate parasites of various forms and dimensions. Some are in process of division. All possess a voluminous nucleus.

5. Forms described as encysted have the size of a red cell, show two chromatin masses in their cytoplasm and numerous chromophile granulations. In other cases, nuclei of various forms and shapes occur.



Franchini has never found forms of the ordinary *Leishmania* type either free or enclosed in leucocytes. Professor LAVERAN has examined the preparations and considers the flagellates (herpetomonads) distinct from trypanosomes.

H. B. F.

BRUMPT (E.). **A propos de l'*Haemocystozoön brasiliense* de Franchini.**—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 377-380. With 3 figs.

Brumpt has examined one smear of *Haemocystozoön brasiliense*, from the blood of FRANCHINI's patient (see this *Bulletin*, Vol. 2, p. 45) taken some days before death. He has seen all the forms described by FRANCHINI, flagellates excepted. He considers that the name *Haemocystozoön* should disappear and that the organism should be called *Herpetomonas brasiliense*. The non-flagellate forms of the organism are compared with those of *Trypanosoma cruzi* and those of the flagellate of *Melophagus ovinus*. Brumpt suggested that the development of flagellate forms and of cysts was due to contamination of the preparation outside the body of the host, especially as bacteria were present. This idea was combated by LAVERAN (in whose laboratory FRANCHINI works) who stated that bacteria were present as a very exceptional circumstance in the numerous preparations made by FRANCHINI and hence inferences could not be drawn from the one preparation examined by Brumpt.

A. P.

CHATTON (Edouard). **L'Ordre, la Succession et l'Importance Relative des Stades, dans l'Evolution des Trypanosomides, chez les Insectes.**—*Compt. Rend. Soc. Biol.* 1913. June 6. Vol. 74. No. 20. pp. 1145-1147.

According to the author, in the Trypanosomidae of Insects (series *Leptomonas Crithidia*) the order, succession and characters of the stages are as follows:

1. Monadine stages—flagellate, with blepharoplast anterior to nucleus, and capable of division.

2. Trypanoid stages (in the case of *Leptomonas*) or trypanosomes (in the case of *Crithidia*). Derived from the former by retrogression of the blepharoplast; usually incapable of division; stages of "genetic repose."

3. Gregarine-like phases—monadines which have absorbed their flagella. Immobile, fixed anteriorly to intestinal cells. Form agglomeration rosettes, being capable of division. This is not a stage but an adaptive form.

4. Spermoid stages. Derived from the last-mentioned by retrogression of the blepharoplast. Repose forms, incapable of division. Nucleus and blepharoplast near together. They become

5. Encysted forms, with eosinophile capsule.

Such a complete succession is "diphasic." Simplifications often occur, such as the suppression of the trypanoid or trypanosome stages, when the evolution is said to be "monophasic."

The essential stages are the monadine and spermoid stages.

H. B. F.

MARTIN (C. H.). **Some Remarks on the Behaviour of the Kinetonucleus in the Division of Flagellates: With a Note on *Prowazekia terricola*, a new Flagellate from Sick Soil.** — *Zoologischer Anzeiger*. 1912. March 14. Vol. 41. No. 10. pp. 452-456. With 8 text-figs.

The author first reviews the literature relating to the division of the kinetonucleus or blepharoplast of trypanosomes and trypanoplasms. His own researches on *Trypanoplasma congeri* gave no indication of mitotic division of this organella.

*Prowazekia terricola* was found in some cultures of a sick soil. It is spindle shaped, measuring  $12\mu$  by  $6\mu$ . No trace of mitotic division has been found in the kinetonucleus. On the contrary, the kinetonucleus swells and is simply constructed into two. Encystment occurs.

The author remarks that whether the kinetonucleus in flagellates is to be regarded as a true nucleus or not is still to him an open question, but that WERBITZKI's work has shown that the old idea of the kinetonucleus regulating the organs of movement is no longer tenable.

The text-figures illustrate the morphological details described in the paper.

H. B. F.

MARTIN (C. H.). **Further Observations on the Intestinal Trypanoplasmas of Fishes, with a Note on the Division of *Trypanoplasma cyprini* in the Crop of a Leech.**—*Quart. Jl. Microsc. Science*. 1913. May. Vol. 59. Pt. 1. (New Ser. No. 233). pp. 175-195. With 2 plates and 2 text-figs.

The author has reinvestigated the flagellate from the stomach of *Box boops*, previously described by LÉGER as *Trypanoplasma intestinalis*, also the flagellate from the stomach of *Cyclopterus lumpus*, and *Trypanoplasma congeri*.

*Trypanoplasma congeri* shows in normal multiplication, division of the basal granules, splitting first of the anterior flagellum and then of the posterior flagellum and membrane, followed by nuclear division and cytoplasmic cleavage. Abnormal division forms have been found, in which the kinetic nucleus only enters one daughter organism, while the main nucleus, though showing division and ultimately forming two nuclei, remains in the second daughter form. The first of the two forms degenerates; the fate of the second is unknown. Rounding-up forms, similar to those found in some trypanosomes, are also described.

*Heteromita dahlui*, Apstein, = *Trypanoplasma ventricoli*, Keysseltz, = *Diplomastix dahlui*, Mobius, occurs in *Cyclopterus lumpus*. The organism has marked euglenoid movements. A mouth is present. It has two flagella, the anterior one being much thickened at its base and the posterior one being closely in contact with the body so that no undulating membrane is present. The nuclei vary in appearance. Longitudinal division is described in detail. The parasites are restricted to the stomach of the fish and are immobilised quickly by intestinal juice.

*Trypanoplasmoïdes intestinalis* = *Trypanoplasma intestinalis*, Léger, = *Cryptobia intestinalis*, Alexieff, occurs in the stomach of *Box boops*. It shows three flagella in life very clearly. The organism is "roughly carrot shaped," three free flagella originate from its blunter extremity, the fourth turns along the body and ends freely. Division is described, which occurs in the smaller or medium sized forms. Rounded forms, possibly encysting, also are present. Because of its movements, method of division and three anterior flagella, the organism is placed in the genus *Trypanoplasmoïdes*.

The division of *Trypanoplasma cyprini* in the crop of a leech was seen, chiefly in the later stages.

The author concludes with a comment on the nomenclature of the bimastigote flagellates, the need for a re-investigation of the *Cryptobia* of snails and a protest against the undue importance assigned to the kinetic nucleus which is not necessarily homologous in all flagellates.

A. P.

FRANCHINI (G.). Sur un Protozoaire nouveau parasite de *Anopheles maculipennis*.—*Compt. Rend. Soc. Biol.* 1913. June 13. Vol. 74. No. 21. pp. 1196-1198. With 18 text-figs.

The new parasite is rare, having been found in one mosquito out of several hundreds examined. It is described under three different "aspects, thus:—

1. Free parasites, round or oval, about  $3.5\mu$  in diameter, and uninucleate. They are unpigmented.
2. Multiplicative forms, encysted, measuring  $8.3\mu$  to  $9.9\mu$  in diameter, and dividing by a series of bipartitions into eight daughter forms. The latter are ovoid measuring  $3.5\mu$  by  $2.5\mu$ .
3. Encysted forms, round or oval, measuring  $4.5\mu$  to  $5\mu$  by  $2.5\mu$ , with thick cyst wall and large nucleus.

Franchini considers the parasite to be new, and its development to be completed within the digestive tube of *Anopheles maculipennis*. He names the organism *Maccellie anophelie*, n.g., n. sp.

The paper is illustrated by 18 text figures.

H. B. F.

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## BOOK REVIEWS.

**EHRlich (Paul).** *Abhandlungen über Salvarsan.* Vol. 3. 584 pp. With 2 plates and 49 text-figs. 1913. Munich: J. F. Lehmann's Verlag. [Price, paper 10 Mk.; cloth 12 Mk.]

This work consists, like the previous two volumes, of reprints of papers published, chiefly in the *Munchener Medizinische Wochenschrift*, on salvarsan and neosalvarsan. There are Sections on Technique (the "water error" and saline fever), The use of salvarsan in Navies and Armies, Abortive treatment of syphilis, Reinfection after treatment, Nerve relapses and Lumbar puncture, Secondary Effects and Deaths, Salvarsan in Non-Syphilitic Diseases (scarlatina, noma, chorea, etc.), Salvarsan in Exotic Diseases, and Neosalvarsan. Articles by Ehrlich introduce and close the volume.

All is of interest to workers in the tropics; the last two sections more particularly concern the readers of this *Bulletin*. Marine-stabsarzt Dr. G. ROST writes on Salvarsan in Yaws (pp. 409-413). The cases were seen in the Yaws Hospital at St. Augustine, Trinidad, under the care of Dr. ALSTON. The salvarsan was given as oil emulsion intramuscularly. Intravenous injections were excluded owing to the lack of the necessary apparatus. The oil emulsion is a simple means of administration. Oil is a bad medium for bacteria, so that there is slight danger of sepsis. There were about 1,000 cases treated; 500 are here considered. The first was treated in January and the last in October, 1911. The average dose was 0.6 gm. for adults, for children up to 0.2 gm. At the end of November 1911 498 cases were cured, that is to say 99.6 per cent., 409 after one injection, 75 after two, and 14 after three injections. Only two cases had failed to be cured and received a third injection. Only four relapses were seen, all of them after one injection; these cases were cured by a second. No complications attributable to the drug were seen, though 75 per cent. of the treated were children. The author thinks that this method of administration is specially suitable in tropical practice for expeditions and in the field.

The work of CARYOPHYLLIS and SOTIRIADES on the treatment of kala azar has been noticed in this *Bulletin*, Vol. 1, p. 6, as that of v. PETERSON on the treatment of Tropical Sore (*loc cit.*, p. 372), and that of HATA on treatment of Rat Bite Disease in Japan (*loc cit.*, p. 407).

IVERSEN and TUSCHINSKY write on the treatment of tertian malaria with neosalvarsan at St. Petersburg (pp. 497-502). Five cases were treated. Four had previously had quinine. The temperature charts of three are given. The dose was 0.6 gm. to 0.75 gm. given intravenously. They cannot say whether the disappearance of the parasites is permanent. They prefer to give a second dose ten days after the fall of temperature. They consider neosalvarsan to be a specific for benign tertian malaria.

WERNER's paper on Neosalvarsan in Malaria was summarised in this *Bulletin*, Vol. 1, p. 120.

A list of 132 references is appended.

A. G. B.

CASTELLANI (Aldo) & CHALMERS (Albert J.). *Manual of Tropical Medicine*. Second Edition. xxxii+1747 pp. With 630 illustrations in text. 1913. London: Baillière, Tindall & Cox. [21s. net.]

Published first in April 1910 a second edition of the above work has now appeared. The subject has advanced considerably in that time and an increase of 500 pages has been necessary to deal adequately with the new material. Though no discoveries of a very startling nature nor of great magnitude have been made in the interval yet there have been many smaller ones, and, as already mentioned, the general advance has been a progressive one. With so many nationalities now actively engaged in the study of Tropical Medicine this is not to be wondered at, and a book on the subject, to keep up to date, really requires a new edition at least every three years.

The general arrangement of the new edition follows that of the old, and this is now so well known that it need not be mentioned again here.

The authors have incorporated everything of importance that has happened right up to the moment of going to press, though some of this has not had time to be confirmed and will in all probability require considerable modification in the future. In addition to this insertion of new matter portions of the old text have been corrected where necessary, and everything brought thoroughly up to date. As a text book and as a general book of reference there is no doubt that the manual takes a foremost place amongst the works of tropical medicine of the present day, and the amount of hard work the authors must have accomplished will be thoroughly appreciated by everyone who has a first hand knowledge of the subject. As others have already noticed and pointed out, the index to the general contents and to authors' names contains several mistakes, and it certainly will repay the writers to have this more closely attended to in future editions. Another complaint one has heard is that the size of the book is now too great. Against that however must be put the great advantage of having all one's information in one volume; a great desideratum when moving about and far removed from libraries.

The manual is specially well illustrated, many original drawings, diagrams, photographs and coloured plates being found within its pages, for a number of which the authors are indebted to Signor TERZI, so well known for his skill in delineating insects and other tropical parasites.

G. C. L.

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## TROPICAL DISEASES BUREAU.

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## PLAGUE.

Tonn (P. J.). **A Study of Plague.**—*China Med. Jl.* 1913. Jan. Vol. 27. No. 1. pp. 10-20; and May. No. 3. pp. 158-167.

In order to stay the plague at Macao in 1912, the Chinese paraded the streets at night with lanterns and torches, amid the beating of gongs and discharge of fireworks, so that the evil demons might be terrified and expelled. 1710 people died of plague in Hongkong in that year. Several hundred cases occurred in Canton and many more in the neighbouring villages.

In the author's ten years' experience of plague, the first symptom is often a chill; headache and pains in the bones, nausea and vomiting are frequent during the twenty-four hours following the onset. The fever in severe cases mounts to 104°-106° F., and at the end of the second or the beginning of the third day it falls to 102°-103°. By the end of the third day or on the fourth day it reascends to 105°-106° F. when death takes place.

Many cases present atypical symptoms at the beginning and end of an epidemic.

Seventeen out of 40 cases of bubonic plague which were treated with combined intravenous and subcutaneous injections of 20-400 cc. of anti-plague serum recovered. Before the author began serum therapy, he lost all his plague patients. He recommends the administration of ten grains of urotropin every hour until irritation of the bladder is complained of, and the injection of 10-20 drops of pure phenol into each gland, although he quotes KLEIN who found that 1 in 30 formalin failed to kill the *B. pestis* in 15 minutes, and that 1 in 80 phenol must act on the bacillus for 10 minutes before it is destroyed. Digitalis and strophanthus are more effective than strychnine in sustaining the action of the heart.\* Morphine may be employed but antipyrine is to be avoided.

C. Birt.

DE SOUSA JUNIOR (Antonio). *Algumas considerações sobre a Peste Bubonica.* [Some Considerations on Bubonic Plague.]—*Medicina Contemporanea.* 1913. June 15. Vol. 31. No. 24. pp. 185-187.

Plague appeared at Oporto in 1899 since when the author has accumulated a few facts relating to it. Recently his work has been carried on in Lisbon.

The author agrees with the German and Austrian commissioners in denying the existence of primary septicaemic plague. The Austrian commission believed that slight generalized plague adenitis was caused by infection from the tonsils as from a primary bubo. The author's cases of plague of the tonsil and fauces did not support this view. He considers his faucial cases in two groups, first those infected by fleas. In 80 per cent. of these the cervical pain and tumefaction preceded the angina, the interval between these varying from a few hours to two or three days. In 20 per cent. of the cases the two appeared simultaneously but in no case did the angina precede the pain and swelling of the neck. At autopsy he found the buboes in the neck haemorrhagic and necrotic and their lesions in no way less advanced than those of the tonsils and fauces. These considerations make him conclude therefore that the fauces and tonsils were secondarily infected from the cervical buboes. In the case of those infected from pneumonic or faucial cases there may be localized faucial attacks which clear up, the sputum never showing many *B. pestis*. The infection on the other hand may reach the lung. In these cases the sputum is loaded with plague bacilli and five or six days after infection cervical buboes appear. These after death are found to be tumid, soft, pale and without surrounding reaction. He only saw one case of primary bubo of the neck from faucial infection in 800 patients. The experience of TERJINAMIA in the case of the Manchurian epidemic of pneumonic plague was similar. He agrees with the view of the advisory committee. "No primary infection of the blood in plague except in the foetus." He states further that slight generalized plague adenitis always starts from a primary bubo, which careful examination will reveal. It does not start from the tonsil.

W. J. Penfold.\*

HARKER (W. E.). *Outbreak of Plague amongst Apprentices on board the British Steamer "Bellaisa."*—*Annual Report of the Medical Officer of Health, Tyne Port Sanitary Authority,* 1912. 1913. p. 24-26.

The s.s. Bellaisa left Rosario on July 2nd, 1912, with a cargo of maize, called at Monte Video on July 18, St. Vincent August 4, Teneriffe August 16, and arrived at Hamburg on August 26. R. H. Morton, one of four ship-apprentices, was attacked with a sudden illness, and died on September 4. The Port Medical Officer of Hamburg, Dr. Sannemann, reported that plague was

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\* It has been arranged that, from July 1st, the Plague Section shall be in charge of Dr. J. HENDERSON-SMITH. Dr. HENDERSON-SMITH has since gone abroad for three months, and in his absence Dr. W. J. PENFOLD is acting for him.

the cause of death. On September 8 another apprentice, J. R. McWhirter, fell ill. He was put ashore at Cuxhaven where he too died of bubonic plague.

The vessel reached the Tyne on September 10, when a third apprentice, J. W. Raeburn, was seized with high fever, and great prostration. The fauces were congested and there was slight enlargement of the deep lymphatic glands at the angle of the jaw. One cc. of blood-stained fluid was obtained on puncture at this site, from which the *B. pestis* was grown. On September 12 signs of double pneumonia were observed. He died on September 16.

On September 25 after the Bellailsa had been fumigated, 12 rats were collected, one of which was infected with plague.

The three apprentices who died of plague, bought a tame rabbit at Rosario. The rabbit-hutch was situated near the spot where the plague-rat was found. It is supposed that the rabbit became infected by the rat, which was attracted to its cage by the food, for the rabbit died suddenly on the day before the first case of plague occurred, and its body was thrown overboard. The three boys were in the habit of playing with the rabbit in their berth. The four apprentices were quartered in the after part of the ship by themselves. The three victims occupied a large berth on the port side, and the fourth apprentice who escaped, a similar berth on the starboard side. This youth did not interest himself in the pet rabbit. None of the rest of the crew, 35 in number, was attacked.

C. B.

**AUMANN.** Erfahrungen bei einigen in das Hamburger Staatsgebiet eingeschleppten Fällen von menschlicher Pesterkrankung. [Observations on Plague Cases imported into the Hamburg District.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. June 21. Vol. 69. No. 5-6. pp. 353-378. With 5 text-figs.

No plague had been imported into Hamburg since the year 1907, until September 1912 when the s.s. Bellailsa arrived. A full clinical history of the second case of plague (McWhirter) which arose in that vessel is given, and a long account of the bacteriological investigations is appended. The youth was treated with salvarsan, the failure of which has been reported already (see this *Bulletin*, Vol. 1, p. 316).

Ten rats were captured while the ship was unloading at Hamburg, but none were infected with plague.

C. B.

**SIGNORELLI (E.).** Sulle Alterazioni Anatomico-Patologiche che il Bacillo della Peste o la sua Tossina produce nei Polmoni. [Morbid changes produced by the *B. pestis* and its Toxin in the lungs.]—*Lo Sperimentale*. 1913. May 23. Vol. 67. No. 2. pp. 155-166. With 1 plate.

The most noticeable features found on microscopical examination of the lungs of a case of pneumonic plague were, thrombosis of the interalveolar capillaries; desquamation of the alveolar



epithelium; exudation into the alveoli in which the fibrin was not abundant, nor was it of haemorrhagic character except where the capillaries had burst; invasion of the tissues by the *B. pestis*, where it occurred in large clumps.

Five to ten cc. of a solution of the nucleoprotein of the plague bacillus containing .002 gm. per cc. were injected into the lungs of six dogs which were destroyed 48 hours later. The changes which resulted resembled those of lobar pneumonia in the stage of red hepatization. The fibrinous exudate consisted of plugs of fibrin which filled the alveoli; entangled in the fibrin were red and white corpuscles in abundance; hyperaemia and oedema of the lung were marked; the alveolar epithelium, which was degenerated, was detached; no bacteria were present. The injection of *B. pestis* nucleoprotein into the lungs of dogs therefore gives rise to aseptic fibrinous inflammation. In the lung lesions caused by *B. pestis* the fibrinous deposit occurs chiefly in the pulmonary capillaries which become blocked in consequence; in the alveoli it is limited to a coarse network of fine filaments. On the other hand in the inflammation set up by the nucleoprotein of *B. pestis*, the fibrinous exudate is more compact and is confined to the alveoli; the capillaries are free. In pneumonic plague the degenerative processes in the alveolar epithelium and leucocytes are more marked than in the lesions caused by the nucleoprotein.

Pneumonic plague is an inflammation of fibrinous type with localization of the fibrin chiefly in the pulmonary capillaries; the fibrin formation arises from the specific clotting action of the nucleoprotein of the *B. pestis*. The thrombosis of the interalveolar capillaries is one of the factors which causes the severity of pneumonic plague.

C. B.

ROBIN (L. V. E.). *Conclusions Générales des Délibérations de la Conférence Internationale de la Peste à Moukden (Avril 1911).* [Traduit de l'Anglais.]—*Arch. de Méd. et Pharm. Navales*. 1913. Mar. Vol. 99. No. 3. pp. 208-217; Apr. No. 4. pp. 285-293; and May. No. 5. pp. 361-374.

A summary of this work appeared in this *Bulletin*, Vol. 1, pp. 217-226 and pp. 308-312.

C. B.

#### PLAGUE IN PORTO RICO.

CREELE (R. H.). i. *Plague Eradication in Porto Rico.*—*Jl. Amer. Med. Assoc.* 1913. May 17. Vol. 60. No. 20. pp. 1527-1532.

ii. *Outbreak and Suppression of Plague in Porto Rico. An Account of the Course of the Epidemic and the Measures employed for its Suppression by the United States Public Health Service.*—*U.S. Public Health Rep.* 1913. May 30. Vol. 28. No. 22. pp. 1050-1070. With a map and 3 plates; and June 6. No. 23. pp. 1121-1149. With 3 plates.

There was an outbreak of plague in San Juan, the population of which is 50,000, between the middle of June and the beginning of September 1912. General insanitary conditions are not the

cause of plague, for the inhabitants of the most squalid part of Puerta de Tierra, where the houses are built on elevated piers on account of the swampy nature of the ground, remained free from the disease.

26,532 *Mus norvegicus*, 7,953 *M. alexandrinus*, 4,514 *M. rattus*, 6,533 *M. musculus* and 258 mongoose were examined between June 23rd 1912 and February 15 1913; 66 rats were infected, of which 37 were *M. norvegicus*; 4 *M. rattus*; 1 *M. alexandrinus*; 2 *M. musculus*; and 22 were unclassified. In only one were found signs of chronic plague. During July and August a considerable number of rodents was examined in which buboes and marked subcutaneous injection were present, and bipolar staining rods were not infrequent in smears. Not much stress however was laid on the microscopical examination, since saprophytes invading the tissues after death often appear as bipolar staining rods, especially if carbol thionine has been used.

The average number of fleas per rat in 1,940 rats examined was 0.64; more than 90 per cent. were *Xenopsylla cheopis*; *Ctenocephalus canis*, and the chicken flea, *Echidnaphaga gallinæ* were also found, 18 of the latter were obtained from six *M. norvegicus*. The numbers varied considerably; 54 fleas were caught on three rats, but no fleas were found on 26 rats. The rats harboured rather more fleas during the month with least rainfall.

For the eradication of the plague, it was required that dwellings should be raised two feet from the ground with the underpinning free, or that they should be surrounded with a concrete wall extending 2 feet below the surface, and 1 foot above the floor level in the case of food stores; doors and windows to be made rat-proof; fowl-houses and pens to be supplied with concrete floors and walls, and to be protected with  $\frac{1}{2}$  inch mesh wire netting. For the poisoning and trapping of rodents 269 trappers and 9,000 traps were employed. Sulphur fumigation and petroleum spraying of infected premises were carried out. In the case of loosely constructed walls in which were numerous burrows, they were protected by a 4 inch coating of concrete extending 2 feet below and 1 foot above the ground level. Pipes, wires, and trees were guarded by galvanised iron collars 2 feet or more in diameter.

#### Summary.

"There were 55 cases of human plague in the Porto Rico epidemic, 10 having occurred at the date of announcement of the disease and 45 afterwards. All were of the bubonic type, with a mortality of 65 per cent.

"Flea infestation in Porto Rico is very low compared with that in other tropical countries. The data are not sufficient to determine if there is a seasonal variation. The species of fleas encountered on rodents were *X. cheopis*, *Echidnaphaga gallinæ*, *Ctenocephalus canis*, and *P. irritans*, with the addition of *Rhynchoprion penetrans* (chigoe) on the human host. The infestation of rats by the chicken flea in San Juan is noteworthy. The decline in rat population as indicated by the weekly catch was marked, and to a large degree was the result of rat-proofing. The rat-infection extended to 4 points outside San Juan; to Carolina, a distance of 14 miles; Caguas 23 miles; Arecibo about 50 miles; and Rio Piedras 3 miles. Only in the first-named village did human cases occur (3). The presumptive evidence points to an extension by infected rats in merchandise. The infection in Rio Piedras might have been carried there by migratory rodents.

"In Porto Rico the *M. norvegicus*, *M. alexandrinus*, and *M. rattus* infest houses, but the former predominates. *M. alexandrinus* is the species chiefly found in rural districts. Sufficient data are not at hand as to the variation of breeding, but it is probable that there is no seasonal fluctuation of this nature among the rodents of Porto Rico."

C. B.

### PLAGUE IN THE PHILIPPINES.

HEISER (Victor G.). **The Outbreak of Plague in Manila during 1912. The Insidious Beginning, with a Discussion of Probable Factors concerned in its Introduction.** — *Philippine Jl. of Science*. Section B., Trop. Med., 1913. Apr. Vol. 8. No. 2. pp. 109-115. With 1 map.

On June 19th, 1912, plague broke out in Manila. For the previous six years the town had been free from human plague with the exception of imported cases, which however had always been intercepted at the quarantine station.

During the previous April three infected ships arrived at Manila. After the time of arrival of the second of these the temperature of every person, on in-coming vessels, was taken, with the result that in the case of the third ship a Chinese passenger was detained because his pulse was 100 and his temperature 39° C.

Seven days after, this patient's temperature fell and indubitable evidence of plague appeared, the patient dying of the disease.

The first actual case arising in Manila was that of a Filipino who resided in the Chinese district, and died after three days' illness. No satisfactory explanation of the infection of this patient was forthcoming. She had not been out of the city for months. She did not live near the water front, and did not associate with anyone who had been recently out of the city. During the next four months straggling cases occurred in different streets, without apparent connection, but three successive cases were all in boys under sixteen.

In October a sharp extension of the epidemic occurred, when it was found that all the new cases were in labourers who worked in one freight station. This extension of the epidemic was preceded by a heavy rat mortality in the warehouse. From the commencement of the epidemic till the end of August, 3,000 rats per month were caught, but not till the 31st of August was a plague-infected rat found amongst them. Up to October 1st only .005 per cent. of the rats examined were infected as against 7 per cent. for example in Hong-Kong.

Heiser gives an account of the sanitary measures employed wherever a human or rat case occurred. These in no way differed from those customary on such occasions. The fumigation of the sewers was carried out with SO<sub>2</sub>. The fumigation was always accompanied by a high rat mortality from the traps and poison which were used regularly. Arsenious acid and rice boiled together 1:5 made the most successful bait.

The rats of the water front remained free from infection up to the time of writing so that it seems most probable that if the disease were introduced by rats it must have been in cargo. The possibility of bed bugs having been the cause of the introduction of the infection is considered and dismissed.

W. J. P.

Goff (A. P.). **Bubonic Plague in Manila.**—*Jl. Amer. Med. Assoc.* 1913. June 28. Vol. 60. No. 26. pp. 2042-2043.

The cause of the epidemic dealt with was obscure but it was presumed to be due to an infected rat getting ashore in cargo from some boat. In the rat-catching operations few infected rats were found, hence there appears little prospect of a serious epidemic.

The cases reported were 55 in number. All except two occurred on the north side of the river. Twenty-two cases, occurring in October, originated from an infected store-house. After cleaning and disinfection no further cases arose in connection with this building. The race, sex and age distributions of the cases are given. The outstanding feature was the large proportion of young people attacked. This it is suggested may have been due to the greater tendency of the young to go barefooted. Smears from buboes, cultures and animal inoculation were practised in the case of all patients admitted to hospital. Febrile cases showing enlarged lymphatic glands were frequently sent in as plague and these occasionally gave rise to difficulty in diagnosis. The laboratory methods were found very useful in doubtful cases, of which two are briefly described.

The femoral glands were usually the first to enlarge. Skin eruptions occurred in two cases only. Three or four cases, though originally of the bubonic type, developed bloody sputum which showed many plague bacilli.

The attendants on these cases escaped infection. The risk of such infection from cases showing secondary lung involvement was held to be slight owing chiefly to the possibility of having the windows constantly open.

Guinea-pigs left in houses from which patients had been taken became infected.

Serum from the government laboratory was used in the cases, but the author is unable to make any positive statement about its value. Twelve and one half per cent. of those attacked recovered.

W. J. P.

Fox (Carroll). **The Plague Outbreak in Iloilo.**—*Philippine Jl. of Science.* Section B., Trop. Med., 1913. Apr. Vol. 8. No. 2. pp. 119-120. With 1 map.

The outbreak in question was small, only eight cases occurring. They were in two foci a considerable distance apart. A Chinaman living in one of these had business connections with the other. Laboratory examinations of the rats caught during the continuance of and after the outbreak failed to show any infection.

That these animals played no part in the outbreak is further suggested by the circumscribed character of the foci and the sudden cessation of cases after treating the houses believed to be infected in such a way as to destroy vermin.

The author attributes the outbreak to bed-bug transmission, commencing with an imported case of human plague. In the early part of the campaign no endeavour was made to secure bed-bugs for inoculation experiments so that no direct proof of the hypothesis was obtained.

W. J. P.

#### HISTORICAL.

JOURDAN (Victor J. P.). **Bubonic Plague: its History and Prevention.**—*Monthly Cyclopaedia & Med. Bull.* 1913. May. Vol. 16. No. 5. (Old series, Vol. 27.), pp. 270-274.

The author, from historical considerations, attributes the distribution of plague between different countries in the eighteenth and nineteenth centuries to the degree of civilisation reached by these respective countries. He discusses the various views as to the cause of plague, and how some of these prevalent amongst the ancients held sway, until about the middle of the Christian era. In the sixteenth century, as anti-plague measures, the Spanish Government forbade attendance at theatres, schools and even churches; they required the plague hospitals to be outside the city and the attendants on the patients had to wear leather suits. Bonfires had to be lighted in the evenings in various parts of the city. Early burial of the dead (within six hours) and burning of their personal effects was also enjoined. These measures were however ineffective. Various fantastic views held by reputable physicians of the sixteenth and seventeenth centuries are discussed. As a result of the great epidemic at Marseilles in 1720 the systematic study of plague from the clinical and preventive sides received a great impetus. Several collaborators at Montpellier wrote a very full two-volume work on the subject.

Many French scientists visited the East to study the disease during the eighteenth century but without much result. In the early part of the nineteenth century various commissions went to the Orient for the same purpose; the one presided over by BALLARD DE MERU advocated the improvement of the hygienic conditions of life and the deeper burial of the dead as preventive measures, and mercury and iodides as medicinal treatment. The history of the demonstration of the parts played by rats and the rat flea in the transmission of the disease is discussed. The author has some pertinent observations to make on the perfunctory way in which ship fumigation for rat destruction is usually carried out and makes suggestions for improvements in the method.

He very reasonably suggests that Medical Officers at Ports should have a thorough knowledge of ship construction in order to be able to direct this work with intelligence.

W. J. P.

**SUDHOFF (Karl).** *Syphilis und Pest in München am Ende des 15. und zu Anfang des 16. Jahrhunderts.* (Eine Urkundenstudie). [Syphilis and Plague in Munich towards the end of the 15th and beginning of the 16th Centuries.] — *München. Med. Wochenschr.* 1913. July 1. Vol. 60. No. 26. pp. 1439-1443.

The sources from which Sudhoff draws his information are the proceedings of the town council from the years 1459 to 1507 and as a check upon these the official finance records for the same period.

The town at the time in question had a population of about 10,000 inhabitants. In February 1474 suspicious cases occurred in the city, while later in the year accounts of works of charity directed to turn away the plague appeared in the official records. On September 29th, 1474, a mass celebrated the cessation of the epidemic.

The account of the proceedings of the council were at this time sparse, but in the bad plague year of 1483 they were much fuller. The disease ceased to rage severely early in 1484, though at this time a note appears to the effect that the mayor is still out of town on account of it. The medical staff of the town stuck to their posts and received their salaries during the whole plague period. The salaries of surgeon, physician, and midwife, as paid by the city, appear regularly in the accounts.

In 1483 apart from pilgrimages and religious exercises in order to remove the plague the following more strictly hygienic regulations were enjoined. (1) Streams had to be kept clean, (2) Excreta had to be regularly removed, (3) The people's bread had to be officially inspected, (4) Noises on the street were suppressed because of their psychic effect, (5) The movements of beggars were regulated. In addition to these arrangements many others were made which Sudhoff suspects had a hygienic origin.

The account of the expenses attendant on the upkeep of plague patients is fully gone into, this including the amounts paid to midwives for opening buboes. The mortality of the epidemic is indirectly obtained from the number of persons buried from the "Bruderhaus" and other considerations.

In 1495 a further slight plague epidemic occurred. The town finance accounts of this year again give a list of payments for the opening of buboes and during the same month an increase in expenses for the disposal of the dead.

W. J. P.

#### RATS AND RAT FLEAS.

**ILVENTO (Arcangelo).** *Hygienische Beobachtungen über Ratten und Pestprophylaxe im Hafen von Palermo (1906-1910).* [Observations on Rats and Plague prevention at Palermo.] — *Arch. f. Schiffs- u. Trop.-Hyg.* 1913. June. Vol. 17. No. 12. pp. 404-413.

A useful aid in the capture of rats is a sticky substance of the nature of birdlime manufactured by the firm of Panchesi in Venice. A layer of about 5 cm. in thickness is spread on strips

of wood, 50 cm. long by 20-25 cm. wide. A bait is placed in the middle. 20 rats have been caught in this way. Rats avoid traps on which they can discern the odour of man or rats. Traps should not be always baited in the same manner, nor set in the same place; they should be covered with straw, sacking, &c.

Since the year 1906 1,599 rats have been caught in the harbour of Palermo, of which 46 per cent. were *M. decumanus*, 53 per cent. *M. rattus*, and 1 per cent. *M. alexandrinus*. Female rats amounted to just over 40 per cent.; 9 per cent. of them were gravid. 527 rats were killed by chloroform, and were subjected to a complete bacteriological examination; none were infected with plague. Small subcutaneous abscesses were found in 12, caused by common pus-producing bacteria. Cysts in the liver due to *Hymenolepis nana* were frequently observed.

122 fleas were taken from 527 rats, of which 65 were *Loemopsylla cheopis*, 41 *Ceratophyllus fasciatus*, and 6 *Ctenopsylla musculi*. The fleas were examined microscopically after they had been rendered transparent by boiling for a few minutes in caustic alkali, and subsequent transfer to glycerine.

C. B.

STRICKLAND (C.). The Bionomics of the Rat-Flea.—*Brit. Med. Jl.* 1913. May 31. p. 1160.

BACOT (A. W.). The Bionomics of the Rat-Flea. [Correspondence.]—*Ibid.* June 14. p. 1299.

NICOLL (see this *Bulletin*, Vol. 1, p. 66) found rat-fleas, *C. fasciatus*, alive in cage sweepings which had been kept in a glass vessel for more than a year. Finding larvae at the end of the experiment he inferred that the fleas had bred in captivity. Strickland observed living rat-fleas, *C. fasciatus*, for 18 months in such refuse, but as he could not discover larvae or pupae, he came to the conclusion that adult rat-fleas can live in rubbish for a year and a half. To ascertain that they were not multiplying in confinement, he removed all the fully grown fleas by introducing a live-bait, and then examined the material for fleas every day for four months without discovering any; hence he thought that there were neither larvae nor pupae in the detritus. The number of fleas in the refuse greatly diminished in the hot weather; had larvae been present in the sweepings, the fleas should have increased, for according to Nicoll the developmental cycle is shortened to ten days, if the atmospheric conditions are warm and moist.

He concludes that the adult flea survives great lengths of time without food.

Bacot states that the egg stage of the rat-flea, *C. fasciatus*, lasts from 4 to 12 days; the active larval stage from 10 to 114 days. The larva may remain in its cocoon for more than 400 days, while the pupal period may go on from 10 to 12 days. After shedding its pupal envelope, the mature flea may remain in its cocoon for three weeks or more. By allowing fleas to bury themselves in sand at a temperature of 45° to 50° F. in a damp cellar he found that they lived without food for 95 days. When the fleas were

placed in a filter paper which was buried in sand, they survived starvation 31 days only.

He infers that the occurrence of adult fleas in breeding cages long after the host has been removed, is to be accounted for by the larval forms remaining within the cocoon.

C. B.

**CREEL (R. H.). The Rat. A Sanitary Menace and an Economic Burden.**—*U.S. Public Health Reps.* 1913. July 4. Vol. 28. No. 27. pp. 1403-1408.

The consideration of the rat from the sanitary standpoint is extremely brief. On the economic side an attempt is made to estimate the loss resulting to the U.S.A. from rats. The basis of the calculation is the rat census and a consideration of the damage which is effected by these rodents. In San Francisco the rat population has been shown by trapping percentages to exceed the human, while in Porto Rico it has been estimated to equal it. Creel believes that the rat population of the U.S.A. is at least equal to the human.

He estimates the daily upkeep per rodent at half a cent and gives many instances of the enormous havoc wrought by rats in the matter of corn, chickens, eggs, &c. The loss to the cane growers in Porto Rico from rats alone has been estimated at the experimental station there as 75,000 dollars per annum.

If in the U.S.A. one half a cent per day be taken as upkeep and the rat population be taken as equal to the human the cost to the country reaches 167,000,000 dollars per annum.

The author also discusses the possibility of rat clearance; the fecundity of the rat is shortly touched upon and the methods of an anti-rat campaign are dealt with, special attention being paid to the means of rendering dwellings rat-proof.

He believes that traps are frequently relatively useless because they are badly placed. They should be somewhat concealed by scattering meal, &c. upon them, while pieces of straw sacking or rubbish can be so arranged as just to leave the mouth free. Only the bait must be available as food, a striking illustration of this point is given.

W. J. P.

**GRUBBS (S. B.) & HOLSENDOR (B. E.). Fumigation of Vessels for the Destruction of Rats.**—*U.S. Public Health Reps.* 1913. June 20. Vol. 28. No. 25. pp. 1266-1274.

The authors believe that sulphur fumigation is still the most suitable method of getting rid of rats in vessels. It is they say efficient, cheap and safe.

They quote in detail the interesting Report of the Board of Health on Plague in New South Wales, dealing with the difficulty experienced in getting rid of rats in the case of the vessel "Innaminka." This is followed by an account of the fumigation of a series of vessels, in which, various points in the technical practice of the authors are indicated. In fumigating they require all dunnage or lumber to be slung up, not resting



on the floor, all pipe casings must be opened, the sheathing of vessels must be well opened up, and all openings admitting rats must be permanently closed. Their illustrative cases show how neglect of these precautions prevented the successful fumigation of vessels, living rats being found in these localities, though the fumigation had been carried out with as much as five times the official amount of sulphur recommended. The difficulty occasioned by the main deck is illustrated by several cases, sails, bins and living quarters being infested when the holds were free from rats. This is important from the fact that rats here are so liable to get ashore from the decks. The authors describe a series of experiments carried out on ship-board in which rats were placed in dunnage, pipe casings, &c., and exposed to sulphur fumes. A further series carried out ashore in a small, wooden, one roomed building is described, which shows that 3 per cent.  $\text{SO}_2$  acting for six and a half hours was required before the experimentally exposed rats were killed.

The authors recommend in conclusion 4.5 per cent.  $\text{SO}_2$  (obtained by using 5 lbs. of sulphur to every 1,000 cubic feet) for the living quarters and engine room, so that seven hours will fully suffice, while for the hold they believe that 3 per cent. for twelve hours is sufficient.

They describe the usual routine practised at San Juan, Porto Rico, which appears to be very thorough. It is carried out by a specially trained fumigating crew of eight persons, and is so organised that it operates quickly and effectively. A complete supervision of every part of the vessel, simultaneous fumigation of empty rooms and holds, exposure of all nooks and crannies and no concessions to protests are the order of the day.

W. J. P.

BERDNIKOW (A. I.). *Einige neue Ergebnisse über die Epidemiologie der Pest. Untersuchungen der Nagetiere der Astrachanschen Steppe.*—[Some New Results in the Epidemiology of Plague. Examination of the Rodents of the Astrachan Steppes.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. June 4. Vol. 69. No. 4. pp. 251-259.

In the neighbourhood of Chutor Romanenko, 85 versts (56 miles) north-east of Rachinka where there had been a sudden outbreak of bubonic plague, and 40 versts south-west of Bykowa, an important mart on the Volga, 136 ground-squirrels, 82 jerboa (*Dipus*), 4 marmots, and 5 field mice were caught.

A ground-squirrel and a jerboa were found to be infected with virulent plague bacilli. In another ground-squirrel signs of chronic plague were discovered, necrotic areas containing virulent plague bacilli being present in the liver and spleen. The animal had been inoculated with the spleen of an apparently healthy jerboa. In all probability the lesions found in the ground-squirrel ante-dated the inoculation.

It seems that there was an epizootic of plague among the ground-squirrels in July which had died out at the end of September when this enquiry was commenced. Signs of acute plague were not

observed in any of the infected rodents. In many animals plague-like bacilli were seen on microscopical examination, but cultures and inoculations failed. The author thinks that these might have been infections with weakened *B. pestis*.

The ectoparasites of the animals were not investigated. He thinks, however, that it is very probable that fleas are the transmitting agents.

C. B.

#### TREATMENT.

LEMAN (Isaac Ivan). **The Treatment of the Plague.**—*Southern Med. J.* 1913. July. Vol. 6. No. 7. pp. 446-448.

The author reviews briefly the methods of active immunity as applied to plague. The production and administration of Haffkines' prophylactic and Pfeiffer's vaccine, Lustig and Gallette's alkaline nucleo-protein solution, Terni and Bandi's peritoneal exudate vaccine and Strong's attenuated living vaccines are considered.

The results of the use of antiplague serum are touched upon and lastly the symptomatological treatment is discussed. The paper is sketchy and does not contain anything new.

W. J. P.

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## LEPROSY.

BAYON (II.). A Critical Review of Recent Experimental Leprosy Research.—*S. African Med. Rec.* 1913. June 14. Vol. 11. No. 11. pp. 201-222. With 4 plates.

References are given to the reports of more than 20 investigators who have attempted to grow the leprosy bacillus. Kedrowsky isolated non-acid-fast bacteria from the nodules of three lepers, passed them through animals, and obtained acid-fast organisms, from which pure cultures were grown. These bear a close resemblance to avian tuberculosis. Multiplication which takes place only on special media at 37° C., is generally apparent in 10-14 days. Bayon considers that Kedrowsky's culture is true leprosy, but that those of most of the other workers are not. He himself isolated from leprous nodules two non-acid-fast organisms which became acid-fast after passage through animals. His cultures are not pathogenic to guinea-pigs or fowls.

In his survey of the experiments in the transmission of leprosy to animals by inoculation of human virus, he regards the results obtained by Melcher and Ortmann, Wesener, Wnouw, and by himself as successful. Metastases of acid-fast bacteria were found in the organs of a small percentage of rabbits which had been inoculated 4-10 months previously with lepromata. Bayon's own experiments are described in this *Bulletin*, Vol. 1, No. 4, p. 196. He concludes that—

“1. Leprosy can under certain circumstances be transmitted to animals, the main difficulties being the high percentage of unsuccesses and the long incubation.

2. The most suitable animals appear to be the rabbit, rat, or mouse. In the rabbit the intraocular method is preferable. The resulting lesions in the inner organs are often not visible to the naked eye, and are similar to those caused by certain strains of human tubercle.

3. In the rat the incubation is also very long, the resulting lesions comparable to those occurring in spontaneous leprosy of the rat.

4. Not all leprous patients' nodules are equally “infective” for animals. In some cases the lepra bacilli appear to be absolutely incapable of multiplication, for reasons we do not at present know.”

Bayon believes that rat leprosy is related to the human infection. He succeeded in isolating from a rat which had been inoculated with a nodule from a leprous rat, an acid-fast rod which resembles avian tubercle in its microscopical and cultural characteristics; but he failed to find this organism in many other leprous rats.

He has used the complement deviation test in distinguishing between the various acid-fast cultures. By using various dilutions of antigen combined with corresponding dilutions of leper serum slight differences may be observed. Kedrowsky's culture when used as antigen deviated the complement in dilutions which failed to do so with Duval's and Rost's bacilli.

\* The test has been applied to the diagnosis of leprosy. It fails in early anaesthetic cases.

Though Leboeuf and others have studied experimentally the question of insect carriers of leprosy, no positive results have been recorded. Prolonged contact between the healthy and the diseased under insanitary conditions seems necessary for leprosy to be transmitted, and then probably not more than 5-10 per cent. become infected.

C. Birt.

FRASER (H.). Report from the Institute for Medical Research [Kuala Lumpur, Federated Malay States] for the period October 1st, 1912, to March 31st, 1913.—Received in Colonial Office, June 30th, 1913.

H. Fraser has continued his attempts to cultivate the *B. leprae* [see this *Bulletin*, Vol. 1, pp. 732-733.] 373 inoculations were made of the bacilli obtained from non-ulcerating nodules of 32 lepers, and the tubes were incubated for periods extending to 9 months, but no multiplication was observed on any occasion. Blood, serum, placenta, salt solution, Ringer's fluid, and milk were among the media employed, aerobically and anaerobically.

Diphtheroid bacilli were isolated but are considered of no importance on account of their ubiquity. He thinks that the investigators who have described the transformation of a non-acid-fast into an acid-fast organism were deceived by transferring unwittingly lepra bacilli along with their saprophytes. He protests against applying the term acid-fast to bacteria which retain the stain only after momentary immersion in weak acid.

Inoculations of leprosy material have been made into 20 animals including a gibbon: no transmission has been observed.

C. B.

#### SYMPTOMATOLOGY.

IMPEY (S. P.). *Symptomatology and Diagnosis of Leprosy*.—*S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 239-245.

After an incubation period of about 2 years tubercular leprosy is ushered in with a febrile attack lasting a few days, which generally recurs several times. Erythematous patches appear on all parts of the body. Most of these soon fade except those on exposed surfaces, some of which become nodular; the scalp, however, escapes. In cold climates the tubercular cases are twice as numerous as the anaesthetic, and the nodules are scattered on most parts of the body and seldom break down; in S. Africa 70 per cent. of lepers suffer from the anaesthetic type, and in the tubercular lepers the nodules are confined to exposed surfaces and generally ulcerate. If the nodules are smooth and soft life is prolonged about 8 years, if they are small and hard 20 or 30 years. In the acute forms there may be sloughing of the infiltrated tissues. The author thinks that it is doubtful whether leprosy attacks the lungs, and that acid-fast bacilli in the lungs of a leper are probably tubercle bacilli. Death is caused most frequently by tuberculosis, or amyloid disease of the kidneys and intestines. Leprosy bacilli appear early in the nasal secretions, but sometimes the examination must be repeated several times before they are detected. Elephantiasis, Madura foot and syphilitic ulcers may be mistaken for tubercular leprosy.

Anaesthetic leprosy begins with severe neuralgic pains throughout the body but especially in the arms, legs, fingers, and toes. Erythematous patches appear, the size of which varies from one to several inches in diameter; some fade. The bacillus attacks the trunks of the ulnar, peroneal, and facial nerves only, but if the case is of more than 4 years standing it is seldom possible to find any leprosy organisms in these nerves. The neuritis never extends to the brain or spinal cord, and does not reach far above the knee and elbow joints. Recovery may be permanent after a mild attack of anaesthetic leprosy: a boy, one of a family of lepers, suffered from pains in his hands and feet, and numbness of his little fingers for 6 months, after which the symptoms disappeared; ten years later he was still in good health. In early cases of anaesthetic leprosy it is impossible to find leprosy bacilli; lead, arsenic, and alcohol cause neuritis which may resemble anaesthetic leprosy. The erythematous patches, and the facial paralysis of leprosy are then of diagnostic importance. In syringo-myelia the fingers are contracted and rigid, but the hand has not the claw-like appearance of the leper hand: the fingers are not rigidly contracted in leprosy. There are no anaesthetic areas in syringo-myelia.

C. B.

WOOD (D. J.). *The Eye Complications of Leprosy.*—*S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 245-246.

In anaesthetic leprosy up to sixty years of age, the eyes are affected in more than half the cases. Ninety per cent. of tubercular lepers suffer from eye complications in the first ten years of the malady; nearly all who survive become blind. In the former type paralysis of the orbicular muscles of the lower lids, epiphora, corneal opacity and those caused by the disease succeed each other. In tubercular leprosy the bacilli attack the lids, conjunctivae, sclerotic, cornea, and iris. Atrophy of the ciliary muscle is an early sign of iridocyclitis which attacks a large proportion of tubercular lepers. In iridocyclitis acute pain is uncommon, but photophobia which is not amenable to treatment may be intense. Iridectomy has no prophylactic action. Treatment is hopeless.

C. B.

MILLER (Thomas). *Early Diagnosis of a Case of Leprosy much assisted by the X Rays.*—*Lancet.* 1913. July 26. p. 219. With 2 text-figs.

A Chinaman aged 22, had noted that his left great toe had been diminishing in size for 2 years. Only an atrophied proximal phalanx could be felt, and there was anaesthesia of the skin over the great toe and the next.

Skiagrams showed loss of the terminal phalanx of the great toe, and partial absorption of the proximal phalanx and of the terminal phalanx of the second left toe. Rarefaction was beginning in the distal phalanges of all the other toes of both feet.

C. B.

LAGANE (L.) & COLOMBIER (P.). **Formule Sanguine de Léproux séjournant en France.**—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 418-423.

The blood counts of eight lepers who were in good health for the most part, varied considerably, hence they are of no aid in the diagnosis or prognosis of leprosy. In the early stages, if the general health is good, there is little anaemia; later this becomes more pronounced, and during the exacerbations of the infection it may be great. There may be leucocytosis or leucopenia. Leucocytosis (14,000, 18,000) was observed both in febrile cases and in early anaesthetic leprosy. The percentage of mononuclears was above the normal in 4, and was normal or below the normal in the other four. In nodular leprosy the medium-sized mononuclears are increased. There was an augmentation in the large mononuclears during a febrile attack (one case) and in two lepers advanced in the course of the disease. The eosinophiles were in the normal ratio or below the normal in 5 of 8 cases. Of the remaining 3 they numbered 5 per cent. in 2, and 10 per cent. in the third which was in the first stage of the infection.

The Wassermann test was positive in 5 of 7 cases and negative in two, one of which was in the early stage of anaesthetic leprosy.

C. B.

#### VARIOUS.

THIBAULT (E.). **De Quelques Recherches sur la Valeur comparée du Mucus nasal, du Suc ganglionnaire, et du Sang pour le Diagnostic de la Lèpre.**—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. June. Vol. 4. No. 6. pp. 293-298.

According to Marchoux rat-leprosy bacilli may be found in the superficial glands of healthy rats. Leboeuf discovered leprosy bacilli in the glands of an early case of anaesthetic leprosy, but failed to do so in the glands of four healthy contacts. Sorel obtained positive results on examination of the glands of 8 out of 19 lepers, and of 2 out of 15 contacts.

Thibault examined the nasal secretion provoked by iodides, the glands, and the blood of 30 lepers; the leprosy bacillus was found in the nasal mucus of 20, in the glands of 18, and in the blood of 7. In no case was it found in the gland when it was not detected in the nasal discharge.

C. B.

SUGAI & MONONOBE. **The Examination of Lepra Bacillus in Circulating Blood of the New Borns.**—*Sei-i-Kwai Med. Jl.* 1913. July 10. Vol. 32. No. 7. (Whole No. 377.) pp. 102-103. (Original in *Jl. Tokyo Med. Assoc.* 1913. Vol. 27. No. 8.)

Leprosy bacilli were found in the placenta of 2 out of 5 mothers who were suffering from anaesthetic leprosy, lepra cells were present in two. Bacilli were discovered in two out of seven placentas in tubercular leprosy, and lepra cells in four. Leprosy

bacilli were present in the blood of 10 out of 12 of the newly born offsprings of these lepers, and in the blood of a seventeen month-old child of an infected mother, which died of pneumonia.

C. B.

#### TREATMENT.

DAVIES (Thos. Sydney). *Notes on the Specific Treatment of Leprosy by means of a Cultural Extract.*—*S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 247-248.

A European girl aged 8, had suffered from dusky red maculae on her face, trunk and limbs for 9 months, received 17 injections of an extract made from Bayon's bacillus in the course of 3 months. The maculae became red and inflamed a few hours after the injections, but soon improved. Six months later those on the body and limbs were almost invisible, but those on the face persisted, although they had faded to a great extent. The remedy was tried on six other lepers, but the results are not reported.

C. B.

VEILLON (A.) & LAGANE (L.). *Action défavorable de l'Arsénobenzol dans la Lèpre.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 415-417.

The authors quote Jeanselme who collected the results of many observers on the action of salvarsan in leprosy. He concluded that it has no beneficial effect. Monte-Santo, however, claims to have cured one case out of four to which the remedy was administered. The authors gave five intravenous injections of 0.3-0.6 gram of salvarsan to a man who had been suffering from the mixed type of leprosy for 3 years. Six days after the last dose he was seized with severe pains in his legs; the skin became greatly infiltrated, and he was much reduced by fever which continued for six weeks. Although some of the infiltration into the skin disappeared, the leprosy lesions advanced rapidly. The Wassermann reaction remained positive before and after the injections. A woman afflicted with mixed leprosy for 5 years received two intravenous injections of 0.25 and 0.2 g. of salvarsan. This was followed by fever which lasted two and a half months, and by solid oedema of the face and limbs, which subsided partially, but the leprous processes appear to have been aggravated. The Wassermann reaction was negative throughout. Five doses of 0.5 g. were given to a case of anaesthetic leprosy of 2 years' standing; no reaction or change in the condition was noted. The Wassermann test was negative.

C. B.

HEYMANS (A.). *Surgical Treatment of some Eye Affections in Lepers.* *S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 246-247.

The most common eye affection is lagophthalmos, which soon causes epiphora and corneal ulcer. This may heal under atropine, iodoform, aristol, and dionine, but relapses are frequent. To

remedy the lagophthalmos the author dissects a flap of skin from the outer third of the lower eyelid, and stitches it to a corresponding area denuded of skin in the upper eyelid. The lower paralysed eyelid is supported by this means. It may be necessary to carry out a similar operation on the inner third of the lower eyelid. Corneal ulcers may perforate and set up iritis which leads to blindness. Many cases of iritis in lepers resemble that seen in syphilis.

C. B.

SANDES (T. Lindsay). *The Surgery of Leprosy.*—*S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 229-233.

The administration of general anaesthesia requires great caution, local anaesthesia with 2 per cent. novocaine is to be preferred, or spinal analgesia with stovaine. The operation area should be washed, swabbed with alcohol, and painted with 1 per cent. alcoholic solution of iodine 24 hours before the operation, when the process is repeated. Strong antiseptics are to be avoided; creolin hycol, and hysol are suitable for ward use. Chromicised catgut is better than silk for buried sutures.

Syphilis is found in about 8 per cent. of lepers. The author gave intravenously 7 or 8 full doses of salvarsan to lepers without observable effect.

Tuberculosis of the lungs, glands, spleen, liver, kidneys, joints, peritoneum, testes, or meninges was noted in 50 per cent. of leper autopsies.

Only two cases of malignant disease occurred in 600 lepers in the course of two years.

A leper was given 239 grains of lead salts over a period of 6 months before lead poisoning came on, manifested by permanent deafness and temporary epilepsy.

Nearly all lepers suffer from ulcers; rest is often essential; skin grafting may be required. Operations for the removal of necrosed bone in the hands and feet are frequent. Necrosis of the nasal septum rarely leads to infection of the adjoining sinuses.

Excision of the thickened portion of the ulnar nerve aggravated the symptoms. Tracheotomy is indispensable in the laryngeal obstruction of late nodular leprosy. Atrophy of the testes constantly occurs in European leprosy.

Iridocyclitis is very chronic. A delay of one or two years is advisable after its subsidence before an iridectomy and lens extraction are performed; the results are often bad. Plastic operations for the relief of ectropion which leads to corneal ulceration may be necessary.

C. B.

MORROW (R.). *The Care of the Leper.*—*S. African Med. Rec.* 1913. June 28. Vol. 11. No. 12. pp. 234-239.

Leprosy is spreading in parts of South Africa; in 1895 there were 148 lepers in Basutoland, in 1912, 700. It is probable there are 4,000 lepers, almost exclusively natives, in British S. Africa, of whom a large proportion is at large. Nodular and



anaesthetic cases should be segregated separately; those infected with tubercle should be isolated. The segregation of lepers is necessary, but their surroundings should be as comfortable as possible, and they should be allowed congenial occupations and amusements; moreover fewer restrictions need be placed on long-standing anaesthetic cases, in which it is probable that the lepra bacilli have died. Robben Island where many of the Cape Colony lepers are segregated, is a barren and bleak storm-swept island two miles in length, and less in breadth. The frequent fogs and absence of vegetation make it an inhospitable spot, residence in which adds to the misery of the leper.

C. B.

## LEPROSY IN RATS.

RIDLON (J. R.). *Note on Leprosy in Rats.*—*U.S. Public Health Reps.* 1913. July 11. Vol. 28. No. 28. pp. 1447-1448.

In the last quarter of 1912 5,700 rats were examined at Mayaguez, Porto Rico; in three, subcutaneous nodules, some of which had ulcerated through the skin, enlarged axillary glands, and patches of alopecia were present. Acid-fast bacilli were numerous in the skin lesions and in the swollen glands, but none were discovered in the viscera. There is human leprosy on the island.

C. B.

LEBOEUF (A.) & SALOMON. *Note sur la Lèpre des Rats en Nouvelle-Calédonie.*—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 484-485.

Rat-leprosy exists in two forms in New Caledonia; the more common is the glandular; four in 136 rats examined were thus affected. Much rarer is the musculo-cutaneous and visceral infection, for one instance only was discovered in 307 rats inspected. In this animal, the loss of hair in patches, the nodules in the skin, many of which had ulcerated, the great enlargement of the lymph glands and the nodules in the liver and lungs completed the picture of this type of the disease. Rat leprosy bacilli were present in all parts of the body.

C. B.



## BERIBERI.

## ETIOLOGY.

EIJKMAN (C.). Ueber die Ursache der Beriberikrankheit. [On the Causation of Beriberi.]—*Munchen Med. Wochenschr.* 1913. Apr. 22. Vol. 60. No. 16. pp. 871-872.

In a short note the author makes a correction of a mis-statement of his views relating to the etiology of beriberi, made by WIELAND in No. 13 of the *Munchener Medizinische Wochenschrifts*, and draws attention to the fact that VORDERMANN's careful observations were not made (as would appear from WIELAND's representation) before, and independently of EIJKMAN's experiments on animals, but after them and with his advice.

P. W. Bassett-Smith.

SCHAUMANN (H.). Zu dem Problem der Beriberi-Aetiologie. ii. [On the Problem of Beriberi.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 13. pp. 433-445.

The author states that he would have preferred to have postponed further remarks upon the above subject until he has terminated the experimental work which he is now carrying out, but he feels compelled to answer some of the objections brought against his previous work. He cannot admit that EIJKMAN has ever taken up a clear standpoint with regard to the present problem, as he always appears to return to the intoxication theory. It is noted that GRIJNS first advanced the partial starvation theory of beriberi, proof of which was established by WIELAND and elaborated by NOCHT and SCHAUMANN. The author points out that two different pathological processes, which depend upon different causes, are in question. The paresis and paralysis, which are easily cured by the antineuritic nitrogen base, appear to be due to a want of "driving energy" for the various nervous processes which can be supplied by the Vitamine; secondly the alterations in the nervous tissue which can only be attributed to metabolic changes (probably in the phosphorus metabolism)—cannot be remedied by the ingestion of an antineuritic phosphorus free nitrogen base. The action of the natural protective substances such as yeast, rice bran etc., depend upon the combined effect of a number of different substances. The antineuritic components cure functional nervous disturbances, but the pathologically demonstrable nervous changes, both chemical and structural, are due to defects in nourishment and a deficiency in phosphorus compounds. In the former there may be no loss of weight, in the latter it is generally marked—both of these conditions are found in beriberi, being most marked in the atrophic forms. In ship beriberi nervous degeneration has only occasionally been observed and these cases are generally rapidly cured by a change of diet containing organic phosphorus compounds, as is also the case in those forms of beriberi attacking well nourished individuals who suffer from paresis without loss of weight or nerve degenerations. The action of nucleo-proteid which Schaumann originally assumed to be the protective body has

received some confirmation from FUNK's experimental work. The reply to FUNK's objection that the body obtained by the author was allantoin, and the priority of the separation of the protective substance, are discussed rather fully—but it is recognised that FUNK was the first to prepare the active nitrogenous base from alcoholic rice extracts. In conclusion the author considers that the very varied results of the many investigators in the preparation of the curative substance proves the uncertainty of the methods as yet used.

P. W. B.-S.

- i. LINDSAY (J. W.). **Etiology of Beri-Beri.** [Memoranda.]—*Brit. Med. Jl.* 1913. July 5. p. 20.
- ii. LAIDLAW (J. D.). **Etiology of Beri-Beri.**—*Ibid.* pp. 20-21.
- iii. FENTON (E. G.). **Etiology of Beri-Beri.**—*Ibid.* 1913. June 14. pp. 1271-72.

(i). The following statements are made: (a) that beriberi occurs in the southern Amazon regions and Matto Grosso in people who have never eaten rice of any kind; (b) that people who have been eaters of polished rice all their lives without any evidence of beriberi, develop the disease when living in the endemic district of Matto Grosso; (c) that cases occur among the agriculturists of Matto Grosso, who eat home-cleaned rice, brown in the grain. From these observations the author is unable to accept the food theory of the etiology of beriberi, but believes that the causative agent is to be found only in the endemic district.

(ii). In the records of the Candelaria hospital, Porto Velho, Brazil, there were 963 cases of beriberi out of 30,430 admissions for the period of 1908-1912. The writer, who contracted the disease, states that rice is very little used and is not causative of the disease, though there may be deficiency in other foods.

(iii). In a short note the author, from ten years' experience in Southern Nigeria, is not prepared to accept the "deficiency theory" as explaining the cause of beriberi. He states that in this happy country young men live as well as in England, the dietary being excellent and varied with practically no polished rice; yet beriberi is found. He asks, what substance is it that is absent?

[The very rosy picture of life in Southern Nigeria is rather different from that usually given. Clinicians generally fail to appreciate that rice is not an essential element in the causation of beriberi, but when, as in the far east, it forms the staple diet the consumption of the polished form is generally followed by the disease. A great monotony of diet and the absence of an essential property (whether from rice, wheat, maize, potatoes etc.) with conditions of life in which metabolic processes are diminished, as from heat, exposure, humidity, and irregular exercise, favour the production of beriberi all over the world. It is probable that the African and American forms vary, both clinically and etiologically, slightly from the typical oriental variety.]

P. W. B.-S.

## CHEMICAL.

COOPER (E. A.). The Preparation from Animal Tissues of a Substance which cures Polyneuritis in Birds induced by Diets of Polished Rice. Part 1.—*Biochemical Jl.* 1913. May. Vol. 7. No. 3. pp. 268-274.

A detailed description is given of analytical experimental work upon the separation of the antineuritic substances effective on pigeons: from which the following conclusions are formulated:— (1) A fraction rich in the antineuritic substance can be precipitated from the fats and lipoids (alcoholic extracts) of various animal tissues by means of ether. (2) A method based on this observation is described for isolating a substance from horse-flesh, small amounts of which can cure polyneuritis in pigeons. (3) The substance is insoluble in absolute alcohol, benzene, chloroform, ether, and ethyl acetate, but it is moderately soluble in water. (4) The substance is absorbed to some extent by animal charcoal and is readily destroyed by alkali. It is not inactivated by sulphuretted hydrogen, but disappears in large amounts during chemical operations in which colloidal metallic sulphides are formed. (5) Quinine and cinchonine exert a temporary curative action upon birds affected with polyneuritis. After being heated at 125 deg. for 6 hours, however, quinine has no ameliorative effect, so that its curative properties would appear to be due to its contamination with traces of the antineuritic substance derived from the cinchona bark. (6) The administration of small doses of alcohol to birds fed on polished rice does not affect the period of time elapsing before the occurrence of polyneuritis, and thus appears not to influence the utilisation of the supply of antineuritic substance distributed in the tissues of the birds. This suggests that alcoholic neuritis does not result from a diminished capacity of the organism to utilise the anti-neuritic substance.

P. W. B.-S.

EIJKMAN (C.). Ueber die Natur und Wirkungsweise der gegen experimentelle Polyneuritis wirksamen Substanzen. [The Nature and Action of Substances efficacious against experimental Polyneuritis.] — *Arch. f. Schiff- u. Trop.-Hyg.* 1913. May. Vol. 17. No. 10. pp. 328-335.

The author reviews critically the modern work which has been carried out bearing on the etiology of polyneuritis of birds and beriberi in man. He fully recognises that FUNK's work is a distinct step forward, but thinks his deductions must be accepted with some reserve, and he states that it still remains to be seen whether "Vitamines" would have the same curative effect on fowls as upon pigeons, seeing that the clinical picture presented by the two birds is different. Dr. van HOOGENHUYZE obtained a crystalline substance from rice bran, which given subcutaneously, had a strong curative action on pigeons fed on polished rice. This substance, at first believed to be identical with FUNK's vitamine, was composed mainly of mineral compounds, the ash giving equally good results—this contained 84 per cent. KCl. Mixtures of

chemically pure KCl and NaCl in the proportion of 3-1 in 20-40 mg. doses gave very good results with pigeons, but even large doses of 300-500 mg. failed to cure fowls. The fact is proved that fowls may be cured by the addition of non-phosphoric compounds to their diet, which contradicts SCHAUMANN's phosphorus starvation theory. SCHAUMANN's latest katalytic theory is discussed very fully, and Eijkman comes to the conclusion that it is probably wrong. He also repudiates the view ascribed to him by SCHAUMANN that beriberi is caused by a poison, and that the protective body acts as an antidote. He says that the poison theory has not been entirely disproved, nor the beneficial action of the protective body as a nerve food been entirely established.

P. W. B.-S.

**BARSICKOW (M.).** *Experimentelle Untersuchungen über die therapeutische Wirkung der Hefe bei der alimentären, multiplen Polyneuritis der Meerschweinchen und Tauben.* [Experimental Study of the Therapeutic Action of Yeast by the Mouth on Multiple Neuritis of Guinea-pigs and Pigeons.] — *Biochemische Zeitschr.* 1913. Vol. 48. No. 5. pp. 418-424. With 1 plate.

There are a great number of preparations on the market made by drying yeast at low temperatures, most of them contain more or less living yeast cells; those known as Furonculine and Levurinose are mixed with a large quantity of starch to better preserve the living cells. "Zymin" is prepared by mixing one part of fresh yeast with fifteen parts of acetone; in this the living yeast cell is killed but the enzyme of the yeast (the Zymase, the Endotrypsin, and the Katalase) remain active. Another preparation is "Cerolin" which is quite different and contains only the fat substances of yeast extracted by hot alcohol. The number of diseases in which yeast has been given therapeutically, both externally and internally, is large, but the beneficial effect has often been much exaggerated especially in skin diseases. The different forms given have no doubt caused variations in the results, therefore it seemed useful to examine the therapeutical effects by the administration of the living cells, and the chemical products only. Chemically there are present, albumin, nuclein, fat, glycogen, carbo-hydrates, water and salts. Four groups of substances were prepared. (1). Carefully air dried fresh yeast containing 70-80 per cent. living cells. (2). Acetone preserved yeast without living cells, but with active enzymes. (3) Cerolin or the fatty substance extracted from yeast. (4) Substances from yeast heated to 120 deg., without living cells or enzymes. To distinguish which of the four groups had the greatest therapeutical value, experiments were carried out with guinea-pigs and pigeons which have been proved by SCHAUMANN and others to suffer from polyneuritis when fed on particular foods, and also to be benefited by being given yeast free from fat. 40 guinea-pigs were divided into 5 groups of 8 in each, and fed upon rice or rice with additions of one of the four above-mentioned yeast preparations; the results were unsatisfactory as all the animals lost their appetite after

8 days and died after 3 weeks. In the second experiment oatmeal was added to the rice, the weight and blood conditions were carefully recorded, but again the results were unsatisfactory—the animals dying in 3 weeks. A third experiment with 40 pigeons was more definite, those fed on *dry* rice plus the yeast preparations all got paralysis early, those having the fat product and the heated yeast died, but the results were different if the rice was given moist, then those only died who received no yeast or only the fat extract of the yeast: the pigeons receiving the heated yeast, the enzyme of yeast, or the living cells plus the enzymes did not die, but no difference was noticed between the birds in the last three groups. The conclusion the author draws from these experiments are that yeast heated to 120° C., yeast with living cells, and yeast without living cells but with enzymes had some protective effect, therefore the protective power of the yeast depends upon some chemical material in the yeast, but he was not able to ascertain whether this was due to the nuclein or the salts.

P. W. B.-S.

FUNK (Casimir). *Studies on Beri-Beri. vii. Chemistry of the Vitamine-Fraction from Yeast and Rice Polishings.*—*Jl. of Physiology*. 1913. June 19. Vol. 46. No. 3. pp. 173-179.

The results of further analytical and experimental work on the anti-neuritic substances contained in yeast and rice polishings are given in detail. In addition to the experiments on pigeons by the author, it is noted that Prof. CUSHNY has shown that these substances possess no marked action on blood pressure, respiration, and the heart. The vitamine-fraction in each instance is found to be complex, consisting of more than one substance. The following conclusions are given:—(1) The vitamine-fraction from yeast has been separated into three substances: a substance of the formula  $C_{24}H_{19}O_9N_3$ , a substance of the formula  $C_{29}H_{23}O_9N_3$  and what appears to be nicotinic acid (m-pyridine-carboxylic acid). The first substance mixed with nicotinic acid seems to be necessary for curing pigeons.

(2) The vitamine-fraction from rice-polishings has up to the present been separated into two substances: one of the formula  $C_{36}H_{20}O_9N_4$  the other nicotinic acid.

WIELAND (H.). *Neuere Forschungen über die Ursache der Beriberi-krankheit.* [Recent Investigations into the Cause of Beriberi Disease.]—*München. Med. Wochenschr.* 1913. Apr. 1. Vol. 60. No. 13. pp. 706-708.

This is a short review of work done by FUNK and SCHAUMANN bearing on the etiology of polyneuritis in birds. It is stated that SUZUCKI, SCHIMAMURA, and OSAKE isolated from rice bran the same curative body, apparently independently of FUNK. The author, though he does not consider the lack of vitamine sufficient to explain the etiology of all cases of beriberi, under which term it is

probable various affections are grouped, yet acknowledges that in many cases the want of the vitamine is the cause of the symptoms. This is shown by the favourable curative results obtained by TSUZUKI when "Antiberiberin," a preparation containing vitamine, was given either by the mouth or subcutaneously.

P. W. B.-S.

#### PROPHYLAXIS.

VEDDER (E. B.). *The Prevention of Beriberi.*—U.S. War Dept. Office of the Surgeon-General. *Bulletin No. 2.* 1913. Jan. pp. 87-94.

Statistics have shown that in the Malay States during the last 20 years 30,000 people have died of beriberi in Government institutions alone. In 1910 there were 3,334 deaths in the Philippines, and it is estimated that 200,000 die annually from this cause alone in China. This is only a small proportion of the total cases as the mortality is about 5 per cent., therefore the total cases must run into millions. There is no doubt that the disease is a preventable one, caused by an improper dietary, which operates because of its deficiency of some substance or substances. The disease has been eradicated from the Japanese Navy and from the Philippine scouts by simple changes in their food; the latter provided 600 cases annually, now there are none. FRASER and STANTON's human experiment is absolutely conclusive, but it has been confirmed by the similar experiment of STRONG and CROWELL in the Philippines. To apply these facts in the far east is difficult, and can only be done by (1) educating the people to demand undermilled rice; such a campaign is doomed to failure, due to the inertia and dislike of oriental people to change their habits; or (2) by taxing over milled rice, making it too expensive for the poor classes. The objections to the latter are, that it would be difficult to enforce, that it would cause native discontent, and that it would derange commerce. The author recommends that International action should be taken, and that a committee of the International Congress of Hygiene should investigate this most important subject.

P. W. B.-S.

HIGHT (H. Campbell). *Studies on Beri-Beri and its Prevention in Siam: being a Report upon certain Investigations on Beri-Beri carried out in Siam by the Medical Officers of the Health Department of the Ministry of Local Government.* 46 pp. With 5 charts. Government of Siam, Ministry of Local Government. Printed by order of H.E. The Minister of Local Government. [Undated.]

This report is a valuable practical exposition of the truth of the "deficiency" theory of beriberi, from experiments made in the public services in Siam. It agrees in all essential respects with the observations made by FRASER and STANTON in the Malay States. The work extended over six years—in asylums, prisons, etc., where all the factors were under control. The author agrees

that the amount of  $P_2O_5$  is a satisfactory indicator (minimum .4 per cent.), and he proves that Siam rice compares favourably both in composition and prophylactic power with other forms on the market; the variations found by FRASER and STANTON being due to differences in milling. He also shows that hand or steam milled Siam rice can be readily prepared and is as efficient as par-boiled rice in preventing the disease. Among other interesting facts given are the following:—In Siam, the incubative period is roughly 60 days; cases are most common in September; and the highest mortality is in young male adults. The author thinks that legislation is not at present desirable, but that white rice should be prohibited in the public services. In the asylum for the insane, which was a hot-bed of beriberi, with a mortality of 90 per cent. of those affected, alteration of the diet from polished to parboiled rice gave the following results:—

Year.			Cases.	Deaths.	Average Daily No. of Inmates.
1906-1907	...	...	116	113	86
1907-1908	...	...	119	104	94.5
1908-1909	...	...	122	122	116.5
1909-1910	...	...	0	3*	193.8
1910-1911	...	...	7	3	310
1911-1912	...	...	14	0	250

\* Cases from previous year.

Parboiled rice was first issued in February, 1909. No other dietic or hygienic changes were made, and beriberi cases mixed freely with the others. This rapid check of the epidemic is very striking, and is in favour of the view that beriberi is not infectious, either from person to person, or from place to place [as found also by STRONG in his Philippine experiment].

P. W. B.-S.



## TROPICAL DISEASES OF THE SKIN.

**SANTAMARIA (J. Martinez).** Some Notes on Tropical Diseases observed in the Republic of Colombia.—*Jl. Trop. Med. & Hyg.* 1913. Apr. 1. Vol. 16. No. 7. pp. 100-102.

In his interesting paper Santamaria mentions several skin diseases as of common occurrence in the Republic of Colombia.

*Buba* (yaws).—This disease is rife in the lower districts while it is never found in the mountainous regions. The *Treponema pertenue* is found in the lesions.

*Bubon de Velez*.—This term is applied to a nodular affection starting near the nose and mouth and somewhat resembling lupus. No surgical or medical treatment is of avail.

*Espundia*.—The author uses this term to denote a condition characterized by pedunculated nodules of unknown origin.

[The same term is often used to denote a type of leishmaniasis affecting the mucosae.]

*Carate* (Pinta).—This is endemic in the hot damp districts and is characterized by the presence of chromatic patches of various colours. **MONTOYA** has, it will be remembered, isolated several species of aspergillus and aspergillus-like organisms from the lesions of this disease.

A. Castellani.

**FLU (P. C.).** Een atypisch geval van *Mycetoma pedis*, gecompliceerd met een blastomycotische infectie. [An Unusual Case of *Mycetoma pedis*, complicated by a Blastomycotic Infection.] —*Geneeskundig. Tijdschr. v. Nederl.-Indië*. 1912. Vol. 52. No. 6. pp. 703-747. With 1 plate.

The patient, an Arab, six years previously struck the great toe of his left foot against a stone, detaching a large part of the nail. Two years elapsed before the wound healed. Some time after this a red nodule appeared in the scar which gradually developed into a tumour involving the whole of the toe and the adjoining parts of the foot. (A photograph of the condition is appended.) The lesion was not painful. The surface was nodular and covered with crusts. Owing to inflammation having set in the growth was removed by operation. Fluid from this showed, microscopically, large cellular bodies exhibiting amoeboid movements, and embedded in these were a number of round bodies about the size of a red blood corpuscle, but more refringent and having a distinct cell wall. Upon section of the tumour it was seen to be studded with grey nodules which, after treatment with caustic soda, resolved themselves into masses of mycelia and spores, the arrangement being more evident in microscopical sections. These researches showed, therefore, that mycelia and blastomycetes were present. Both organisms were cultivated. The streptothrix on agar gave a round, dry, yellow-white mass, strongly adherent to the medium and only capable of being detached in a single piece. After ten days the colour of the colonies changed to a dark brown, probably from drying of the medium. 48 hours growth examined under the microscope showed networks of threads. The organism also grew on maltose

and glycerin agar, on gelatine, on glycerin potato and in broth. On the glycerin potato, in addition to the growths of the streptothrix, colonies of the blastomycete became evident after an interval of three days. These attracted attention by their snowy whiteness and porcelain-like surface. Microscopically, when unstained, the growth showed round greenish-yellow cells from 3 to 6 $\mu$  in diameter, the smaller cells being apparently devoid of a cell membrane but the larger ones presenting a distinct cell wall with double contour. The protoplasm of the smaller cells seemed to be homogenous but in the larger ones a strongly refractive granule could be distinguished. Multiplication took place by fission and by continued adhesion of the cells to one another, chains of as many as thirty individuals being formed. They were difficult to stain, Gram's method showing them up best. A description of the growth of the parasite on various media is given.

Histological examination of the tumour was carried out by means of sections stained in various ways. The nodules took the stain badly, like the caseous matter of tubercle. They ranged in diameter from 0.1 to 2 mm. and high powers indicated that they possessed a definite structure. This consisted of a radiating mass of mycelial threads, terminating at the outer ends in spores, or conidia. The threads stained best by Gram's method but seemed to lose their staining properties by age, so that the centre of the nodules then appeared colourless, the conidia alone retaining the stain. Outside the mass of mycelium and forming the wall of the nodule was a layer of leucocytes of varying thickness, while the bulk of the tumour was made up of granulation tissue. Scattered throughout this were numbers of the large amoeboid cells above mentioned; these contained blastomycetes. Probably these were hypertrophied connective tissue cells, or perhaps they were derived from the cells of the blood vessels. The included blastomycetes, many of which lay free in the tissue, stained moderately well with Gram and similar stains, but best with alum-haematoxylin and eosin. They were, however, most easily demonstrated by treating the fresh tissue with caustic soda solution.

The paper also includes a lengthy discussion of the pathology of Madura-foot, to which class of affection the author considers the present case to belong. In addition, some observations on the occurrence of blastomycetal organisms in tumours are appended.

G. C. Low.

GOUGEROT (H.). *Oosporoses ou Nocardoses Cutanées. Synonymie: Nocardoses, Oosporoses, Oiscomycoses, Micromycoses, Microsiphonoses, Actinomycoses, Streptothricoses.* — *Gaz. des Hôpitaux Civils et Milit.* 1913. Jan. 25. Vol. 86. No. 10. pp. 149-158; and Feb. 1. No. 13. pp. 197-204.

The author gives a full classification of the various pathological conditions due to fungi of the genus *Nocardia*. Some fungi of this genus are of great importance to tropical practitioners as they are found in certain varieties of Madura Foot. The

author calls special attention to the *Nocardia* found in that very interesting condition known as "juxta-articular nodules" characterized by the presence of large subcutaneous nodules in the proximity of the articulations. This disease is common in various parts of the tropics, Madagascar, Java, Samoa, Ceylon, and the Pacific Islands. The etiological factor is a very thin *Nocardia* discovered by Carougeau and the author: *Nocardia carougeau gougérot*. The mycelial filaments are very slender, ramified, without claviform swellings. Attempts to cultivate the parasite have so far not succeeded.

A. C.

DE BEURMANN & GUGEROT. *L'Etat actuel de la Question des Mycoses*. [Rapport présenté au vi<sup>e</sup> Congrès International de Dermatologie et de Syphiligraphie, Rome, Avril 1912.]—Supplement à la *Biologie Médicale*. 69 pp. With 18 text-figs.

This work, which appears as a supplement to the *Biologie Médicale*, is fairly comprehensive as will be seen from a summary of the chapters.

Chapter I.—*Frequency and multiplicity of mycoses*, the chapter contains remarks on the growing number of recognized mycoses, and gives accounts of previous publications on the subject.

Chapter II.—*Sporotrichoses*, deals with the importance of the diagnosis of sporotrichosis in practical medicine, and cites several cases which were wrongly diagnosed as tuberculosis and syphilis. These became steadily worse on being treated with tuberculin and mercury, but when their true nature was determined speedily became cured on the iodo-iodure treatment. This is the longest and most interesting chapter contains the important original work done by the authors on the subject. The rest of the brochure is taken up with chapters on:

Chapter III.—*Botryomycoses*.

„ IV.—*Blastomycoses or Exascoses*.

„ V.—*Oidiomycoses*.

„ VI.—*Oosporoses*.

„ VII.—*Aspergilloses and mucomycoses*.

„ VIII.—*Hemisporoses*.

„ IX.—*Tineae*.

„ X.—*Necessity of International conventions*.

The authors have a good deal to say on the subject of scientific nomenclature and the confusion arising from the use of synonyms. They urge that a revision of scientific nomenclature has now become necessary—especially with regard to blastomycoses—and also that in giving descriptions of parasites, certain technical rules should be followed in order to facilitate the comparison of specimens, as at present descriptions differ widely.

[In the reviewer's opinion every author describing a new species of vegetable or animal parasite should follow the international rules of nomenclature, as set out by BLANCHARD and STILES.]

A. C.

WISE (K. S.) & MINETT (E. P.). *Report on Tropical Diseases Research in the Government Bacteriological Laboratory, British Guiana, for the Six Months April, 1912, to September, 1912.*—Received in Colonial Office, January 21, 1913.

*Aspergillar mycosis.*—The authors describe a case of generalized aspergillar mycosis in a negro woman 55 years of age, who was admitted to the Hospital of Georgetown with high fever, laboured breathing, and patches of dulness on percussion of the chest. She died a few hours later. The post-mortem examination showed at the base of the right lung an area of consolidation which on section had a honeycombed appearance. The heart and spleen were normal. The kidneys contained numerous small cysts. From the lesions the author obtained cultures of a fungus which was determined by FOULERTON as *Aspergillus fumigatus* Fresenius 1775.

[The reviewer has repeatedly called attention to the frequency of bronchomycosis in tropical countries. In Ceylon the condition may be caused by fungi of the genera *Monilia*, *Nocardia*, *Aspergillus*, *Sterigmatocystis*, *Penicillium*, *Mucor*, *Rhizomucor*, *Lichtheimia*, and *Sporotrichum*.]

A. C.

MAZZOLANI (D. A.). *L'Ulcera Fagedenica a Tripoli.*—*Policlinico. Sez. pratica.* 1913. Apr. 27. Vol. 20. No. 17. pp. 585-589; and May 4. No. 18. pp. 621-625. With 2 text-figs.

The author gives a general account of "ulcus tropicum" as observed by him in Tripoli. The clinical characteristics are similar to those described in any text-book. In a long historical account of the researches carried out on the etiology, &c. of the malady, he omits to mention the important work of PROWAZEK. He favours Vincent's fusiform bacillus as the cause of the condition. For treatment Tincture of Iodine is recommended at the very beginning; later caustics may be necessary. No satisfactory results have been obtained by using methylene blue.

[At the present time the prevalent opinion is that the cause of the disease is *Spiroschaudinnia schaudinni* Prowazek 1907.]

A. C.

SABELLA (Pietro). *Due Casi di "Granuloma Ulceroso delle Pudente" guarito col Neo-salvarsan a Tripoli.*—*Policlinico. Sez. med.* 1913. May. Vol. 20. No. 5. pp. 235-240: *Malaria e Malat. d. Paesi Caldi.* 1913. Mar. Vol. 4. No. 2. pp. 97-101.

Two cases of this disease in Tripoli are described, one a man of 35 years and his daughter of seven years of age. The clinical appearances were characteristic in both cases. The little girl's hymen was intact. In both cases spirochaetes were observed, but the author does not come to any definite conclusion concerning the etiology of the malady. Salicylic acid and methylene blue ointments had no effect, while neo-salvarsan given intravenously induced a complete cure in both patients.

A. C.

**CHOYCE (C. C.) & MACCORMAC (H.).** *Case of Granuloma Inguinale Tropicum.*—*Proc. Roy. Soc. of Med.* 1913. Feb. Vol. 6. No. 4. (Dermatological Sect.) pp. 87-88.

A description of a case of Granuloma tropicum in a Chinaman. The first lesions appeared in October, 1911, at the base of the penis from which the disease gradually spread to the left groin and perineal region. Ulcerative and granulomatous changes were evident and there was present a considerable degree of hardening and sclerosis. Wasserramann's reaction was negative. No organisms were found except the usual contaminations. A blood count demonstrated the existence of some anaemia together with a slight degree of eosinophilia. An injection of salvarsan was given without any apparent benefit.

In the discussion that followed SEQUEIRA called attention to a case described by him in which besides the inguinal lesions a granulomatous horse-shoe shaped tumour was present at the angle of the mouth. MACLEOD said that in his experience the best treatment was by X rays. Hayward PINCH mentioned a case in a European which was cured by scraping followed by zinc ionization. McDONAGH speaking on the etiology of the disease reminded the members that WISE had found a Spirochaete and FLU a capsulated intracellular organism in this condition.

A. C.

**GRINDON (Joseph).** *Granuloma Inguinale Tropicum. Report of Three Cases.*—*Jl. of Cutaneous Diseases incl. Syphilis.* 1913. Apr. Vol. 31. No. 4 (whole No. 367), pp. 236-240. With 3 plates.

The disease was first fully described by CONYERS and DANIELS in 1896. Several organisms have been described as the etiological agents: protozoal bodies, and WISE's spirochaete. The author gives a description of three cases seen by him in St. Louis during the last eight years. The clinical symptoms were typical and corresponded to the classical description of CONYERS, DANIELS and MANSON. Cultural experiments showed only the presence of pyogenic cocci. Animal inoculations were unsuccessful. The following conclusions are reached:

- “1. The disease is a rare one in the United States.
2. It is characterized by a striking conformity to type.
3. The adjective *tropicum* as a part of the title of this disease is not wholly justified.
4. The disease in this country is not mainly one of women as MANSON says is the case in the tropics.
5. The negro race is especially liable.
6. The question of contagion must remain open for the present.
7. The peculiar bodies described by Donovan and Carter are not constantly present in this disease.
8. Spirochaetae are not constantly present.”

A. C.

ASSMY & KYRITZ. Ueber Salvarsanbehandlung geschwüriger Prozesse, welche durch die Vincentsche Symbiose veranlasst sind. [Salvarsan Treatment of Ulcerative Processes caused by Vincent's Symbiosis.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Apr. Vol. 17. No. 7. pp. 217-230. With 2 plates and 1 text-fig.

The authors have studied the action of salvarsan in cases of *ulcus tropicum* and *stomatitis gangrenosa* in which *spirochaetes* and Vincent's fusiform bacilli were present. Detailed descriptions of four cases are given. They come to the conclusion that the drug is a specific for such pathological conditions. The paper is illustrated with several excellent photographs, &c.

A. C.

CONYERS-(J. H.). A Case of *Tinea Cruris* in British Guiana.—*British Guiana Med. Annual for 1911.* pp. 16-17.

The author describes a case of *tinea cruris* in a European overseer on a sugar estate. The eruption was cured by the application of an ointment containing gr. xx of chrysophanic acid to the ounce. Scrapings from the lesions were investigated by Dr. MINETT who isolated a fungus closely resembling *Epidermophyton cruris* Castellani 1905 (= *Epidermophyton inguinalis* Sabouraud 1907).

A. C.

## VERRUGA PERUVIANA.

COLE (H. N.). *Verruga Peruviana: its Comparative Histological Study in Man and the Ape.*—*Jl. of Cutaneous Diseases.* 1913. June. Vol. 31. No. 6. pp. 384-392. With 1 plate.

In a previous paper the author describes some inoculation experiments made from verruga sores on to monkeys (see this *Bull.*, Vol. 1, No. 12, p. 726). He now gives the results of a microscopical examination of the tissues then obtained with representations of the growth as seen under low and high powers. In his conclusions it is stated that, (1) No organisms, either bacterial or protozoal were found. (2) The tumours in both man and monkeys resembled each other very closely in their mode of formation, and in their constituents. (3) The tumours are granulomatous in type, caused by some unknown organism, probably circulating in the blood, producing inflammation and obstruction of the lymph channels with sub-acute, inflammatory changes and necrosis. The lymph vessels become choked with mono and polymorphonuclear leucocytes, while around these are seen plasma cells, fibroblasts, mononuclears, and few polynuclear leucocytes. The capillary blood vessels are dilated and there is much extravasation of serum and red blood cells. The lymph vessels either rupture early, or become dilated, their cellular contents degenerate, and the part finally becomes invaded by plasma cells and fibro-blasts.

P. W. Bassett-Smith.

## MISCELLANEOUS.

## MOSQUITOES AND TICKS.

- i. HORNE (J. H.). **Notes on Distribution and Habits of *Stegomyia* Mosquitoes in Madras.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras.* Nov. 18, 19, 20, 1912. pp. 197-199. 1913. Simla: Govt. Central Branch Press.
- ii. LALOR (N. P. O'Gorman). **A Brief Report of the *Stegomyia* Survey in the Principal Ports of Burma.**—*Ibid.* pp. 201-203.
- iii. LISTON (W. Glen). **A *Stegomyia* Survey of the City and Island of Bombay.**—*Ibid.* pp. 187-188.
- iv. MACGILCHRIST (A. C.). **Progress Report—*Stegomyia* Survey—Port of Calcutta.**—*Ibid.* pp. 193-196.
- v. MHASKAR (K. S.). ***Stegomyia* in Karachi.**—*Ibid.* pp. 189-192.

A series of papers dealing with the distribution of *Stegomyia* in different parts of India. An analysis of the work brings out the fact that such insects are common in all the places mentioned. Two species, namely *Stegomyia calopus* (vel *fasciata*) and *Stegomyia scutellaris*, occur in all, with the exception of Karachi where so far the former species only has been met with.

In addition to these, other culicines such as *Scutomyia sugens*, or a species closely related thereto, *Desvoidia obturbans*, *Culex fatigans*, *Culex concolor*, *Taeniorhynchus perturbans*, &c. were found. Some of these seemed to be very frequent. The breeding places of the two species of *Stegomyia* would seem to be very similar, though in Calcutta MacGillchrist did not find *fasciata* breeding in wooden receptacles. *Scutellaris* on the other hand seemed to prefer such places, and hollows in bamboo stumps are also mentioned as favourite sites for it. In the principal ports of Burma, Lalor observed no distinction between the breeding grounds of the two. Most of the reports also deal with the measures required for stamping out these pests.

These are now well known and may be summed up under the heading of general domestic sanitation.

G. C. Low.

HOWLETT (F. M.). ***Stegomyia Fasciata.***—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* p. 205. 1913. Simla Govt. Central Branch Press.

The author in discussing the characteristics of the genus *Stegomyia* states that the plan adopted to check this insect at Pusa has been to eradicate as far as possible all natural and accidental breeding places, but at the same time to supply artificial places (cut joints of bamboo filled with water) which can be emptied out every few days, and so controlled. In the case of all the *Stegomyias* which occur naturally at Pusa (not including *S. fasciata*) the author says that the eggs laid in the cold weather do not hatch until the following rains, lying in earth and dried mud throughout the whole dry season. In those places which



have a well marked cold or dry season the same thing will in all probability be found to happen in the case of *S. fasciata*, as its eggs are known to retain their vitality for weeks or months in a dry condition. In places with an equable warm moist climate these mosquitoes probably breed all the year round. [They do so in the tropics certainly.]

G. C. L.

WILSON (H. C.). **Some Notes on Larvicides and Natural Enemies of Mosquitoes in Southern India.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 183-186. 1913. Simla: Govt. Central Branch Press.

The author believes that for the destruction of mosquito larvae and pupae there is no better natural enemy than fish, other natural enemies of lesser value being found amongst aquatic insects and their larvae, crustacea, molluscs and larvae of non-aquatic insects; the frog should be included as an enemy of the mosquito itself especially when this insect is egg laying or resting on the pupa before its first flight.

Three different fish *Chela argenticia*, *Haplochilus affinis*, and *Ambassis nama* were experimented with. All species of *Chela* are useful but the smaller ones are the best. The class of waters where these species would be of greatest service are tanks, swamps, and village ponds. The genus *Haplochilus*, of which DAY gives four species, are best suited for stocking wells, channels, stagnant pools and any mosquito infested waters at long distances from the breeding grounds. These fish are exceedingly good travellers and if properly prepared for a journey and not over-crowded can be kept in the same water for days together, the water requiring very little aeration.

Another genus of fish, namely *Therapon*, is suitable for stocking back-waters, salt and brackish swamps, and pools near the coast; they can also be used in fresh water ponds.

[More experimental work on such lines would be useful in determining the value of fish as mosquito destroyers.]

G. C. L.

COOLING (L. E.). **Memorandum re Predaceous Habits of Barbados "Millions" *Girardinus poeciloides*.**—MS. Report from Department of Public Health, Queensland. Received in Colonial Office July 1913.

Experiments were made to ascertain the larvivorous propensities and adaptability to environments of the small Barbadian fish *Girardinus poeciloides*.

In the tests applied the fish were found to feed readily on mosquitoes' eggs, larvae, and pupae and also adapted themselves both to large and small bodies of water. The author points out however that before any action is taken to acclimatize them in Australia, notice should be taken of the fact that they are by no means superior to the Queensland fresh water-fishes known as the

Spotted Sunfish—*Rhombatractus fitzroyensis* Castelnau, the Firetail—*Austrogobio galii*, Ogilby, and the Green Perchlet—*Priopis olivaceus*.

[In that case it seems superfluous to go to the expense of introducing the Barbadian species.]

G. C. L.

BISHOPP (F. C.) & WOOD (H. P.). **The Biology of some North American Ticks of the Genus *Dermacentor*. — *Parasitology*. 1913. July. Vol. 6. No. 2. pp. 153-186. With 3 plates.**

In North America more species of the genus *Dermacentor* are found than in any other part of the world, there being at least eight valid species and one variety. The species of the genus may be divided into two groups, based on their habit of moulting on, or off, the host. The group which moults on the hosts is composed of *nitens*, *nigrolineatus* and *albipictus*, that which moults off, of *hunteri*, *parumapertus*, *parumapertus* var. *marginatus*, *venustus*, *occidentalis* and *variabilis*.

Economically this genus is of considerable importance in the United States, as all of the species are of some economic significance except *D. parumapertus*, *D. parumapertus* var. *marginatus* and *D. hunteri*. The first two ticks have not been found to attack any host other than rabbits, and the last has been taken only on the mountain sheep. *D. variabilis* is of little economic importance, so far as known, because among the domestic animals it most commonly attacks the dog. However, it is sometimes taken on the larger animals and occasionally attaches itself to man. *D. occidentalis* and *D. venustus* are common pests of man and nearly all domestic animals, and the latter species, as is well known, is the transmitter of the disease of man called Rocky Mountain spotted fever. The other three species—*nitens*, *albictus* and *nigrolineatus*—are each of considerable importance as live stock pests; none of these are known however to attack man.

A detailed description of all the different species is given and the article is illustrated by three plates.

G. C. L.

#### RAT-BITE DISEASE.

HEWLETT (R. Tanner) & RODMAN (G. H.). **A Case of Rat-Bite Disease.**—*The Practitioner*. 1913. July. Vol. 91. No. 1. pp. 86-87.

The authors describe a case of Rat-Bite Disease which occurred in a London suburb. It was unrecognised at the time but in the light of recent observations was undoubtedly a case of this interesting disease. The patient, a male aged 23 years, was bitten by a rat on the left ring finger at the end of September, 1903. The wound was immediately washed and dusted with iodoform and this dressing was continued until the wound had healed. The wound throughout appeared healthy. About the middle of December a gland at the elbow became enlarged and tender and appeared as if it were about to suppurate. At the end of December the patient had to take to bed owing to the occurrence of rigors

and fever, the pyrexial attacks occurring about every five days for no less than seventeen weeks, when the patient completely recovered.

With the onset of the rigors and fever an erythematous rash appeared on the chest and arms, the glands in the axilla and groin also becoming enlarged. The condition was supposed by some to be tubercular but there were no signs of that disease elsewhere; there was no history of syphilis. Various remedies were tried including quinine, salicylate, arsenic, mercury, iodide, and anti-streptococcic serum, but none of these proved beneficial.

G. C. L.

ATKINSON (A. G.). **Rat-Bite Fever.**—*Med. Chronicle.* 1913. April. Vol. 25. No. 1. pp. 1-28.

The author gives a description of ten cases of Rat-Bite Disease, five of these being hitherto unpublished. He also gives a general description of the disease with references. [For these see this *Bulletin*, Vol. 1, pp. 407-409.] In one of his cases a bite was inflicted by a kitten which had been seen playing with the body of a rat a short time previously. In another case the patient was bitten by a ferret which had just killed a rat. Comparison of these cases with the others leaves no doubt in the author's mind, however, that they were examples of the same disease, the virus being derived indirectly from the rat. In Japan, it is stated, the weasel is known also to be a carrier of the disease. The symptoms in the cases described are typical. [See this *Bulletin*, Vol. 1, p. 407.] The author confirms the observations made by others as to the absence of any parasite in the peripheral blood. [It is possible that the virus may be a filterable one. The fact of so many cases having been reported shows that the disease is by no means uncommon.]

G. C. L.

#### VOMITING SICKNESS.

SCOTT (Government Bacteriologist of Jamaica) writes to say that the statement attributed to him that vomiting sickness is nothing more nor less than fulminating cerebro-spinal meningitis [See this *Bulletin*, Vol. 2, p. 104] is not quite correct. Paragraph 10 of the summary of his Report (omitted from his paper in the *Annals of Tropical Medicine & Parasitology*), explains his views more clearly.

"10. The facts related enable one to say that in a certain proportion of cases exhibiting the Symptom-Complex of Vomiting Sickness the Meningococcus is present, but it does not prove that this is the only cause at work. One is not justified in stating more than this: that, after excluding such conditions as Worms, Gastro-Enteritis, Marasmus, Food poisoning, etc., there is a residue of cases, some of which are due to the Meningococcus, often alone, but at times probably in combination with some other organism, while there still remains an unexplained residue, the cause of which is yet to seek."

G. C. L.

## VARIOUS.

CASTELLANI (Aldo). **A Note on Broncho-oidiosis.**—*Jl. Trop. Med. & Hyg.* 1913. Apr. 1. Vol. 16. No. 7. pp. 102-104.

Remarks on a case of broncho-oidiosis contracted in Ceylon with a relapse in England. The sputum presented microscopically round and oval yeast-like bodies, no tubercle bacilli. On cultivation a fungus was isolated with all the characters of *Monilia tropicalis* Cast. Sajodin gr. xv. thrice daily was administered with good results.

The author makes some general comments on diseases of the bronchi and lungs due to fungi, which he classifies as follows:

1. Those due to fungi of the genera *Monilia*, *Saccharomyces*, and *Cryptococcus*.

2. Those due to fungi of the genus *Nocardia*.

3. Those due to fungi of the genera *Mucor*, *Rhizomucor*, and *Lichtheimia*.

4. Those due to fungi of the genera *Aspergillus*, *Sterigmatocystis* and *Penicillium*.

5. Those due to undetermined fungi.

The symptoms are somewhat similar whatever fungus is the etiological agent. In mild cases there are signs of slight bronchitis with mucopurulent expectoration in which the fungi are found. In severe cases the patient presents the clinical symptoms of phthisis, with hectic fever and haemorrhagic expectoration. As regards treatment potassium iodide is useful in many cases.

G. C. L.

CARSON (G. R.) & CUMMINS (W. T.). **A Case of Coccidioidal Granuloma (California Disease).**—*Jl. Amer. Med. Assoc.* 1913. July 19. Vol. 61. No. 3. pp. 191-192.

Another case of Coccidioidal granuloma is described. The subject was an American clerk living at Coalinga, San Joaquin Valley, California. He was admitted into hospital with severe diarrhoea, headache and a high temperature.

During the last week a cough developed with sweating and mild nocturnal delirium. A clinical diagnosis of typhoid fever was made. Death occurred on March the 2nd, 1913. At autopsy the lesions appeared to be those of miliary tuberculosis, and were largest in the spleen. Microscopically however they were found to be due to the *Oidium coccidioides* (Ophüls). The patient had never presented any skin lesions to account for this infection.

G. C. L.

MARSHALL & MEERWEIN (Wetner). **Über das leukozytäre Blutbild, einschliesslich Verschiebung der Neutrophilen, bei wilden Eingeborenen von Neuguinea.** [On the Leucocyte Blood Picture, particularly with reference to the Neutrophiles in natives of New Guinea.]—*Folia Haematologica.* 1913. Vol. 15. (1. Teil. Archiv.) pp. 229-236.

In differential leucocytes counts, lymphocytosis and eosinophilia were common, the latter probably being due to helminthiasis. A

series of tables with detailed leucocyte counts of different diseases is appended. These include malaria, elephantiasis and other conditions.

G. C. L.

#### ANNUAL REPORTS.

STANLEY (Arthur). **Shanghai Municipal Council Health Department Annual Report, 1912.** 45 pp. 1913. Shanghai: Printed by Kelly & Walsh.

Several infectious diseases of different nature were met with during the year.

*Cholera*: Acute diarrhoea of choleraic type was prevalent from July to September, 1912, the disease affecting both foreigners and Chinese. In none of the numerous cases examined, with the exception of a case introduced from Sungkiang, was the characteristic cholera organism found after repeated and extended examinations. Not only were vibrios of any kind remarkable for their absence from the stools of these cases, but, such as were discovered, showed no agglutination with a specific cholera serum. Though the disease was not recognised as Asiatic cholera the cases were notified as that disease by practitioners and cases conveyed to Japanese ports on ships from Shanghai were declared to be cholera and quarantine restrictions imposed.

*Beriberi*: The incidence of this disease amongst municipal prisoners has diminished. The evidence, according to the author of the report, preponderates in favour of the disease being an infective one, having no direct relationship to food but infective through body vermin. He believes that the recommendations regarding the admission of municipal prisoners will, if carefully carried out, settle the point.

*Dysentery*: Dysentery, with liver abscess as a not infrequent sequel, continued prevalent. The disease as it occurs in Shanghai is mostly amoebic in origin and very prone to produce liver abscess.

*Plague*: Plague-infected rats were found in December 1908, and a complete plague survey of the Settlement has been maintained since. During 1912, 14,988 rats were found dead and brought to the Laboratory for examination, and of these 95 were plague infected, compared with 187, 249, and 138 during the three preceding years. During the year nearly 154,000 rats were trapped and burnt. These, with the rats found dead and examined for plague, brought the total number of rats visibly accounted for to 168,988. In addition to the trapping, close on six million phosphorous baits were laid, about a ton of poison being used. This proved a powerful method in dealing with infected foci. Poisoning on so large a scale carried with it certain risks of course, but after the adoption of poisoned cubes, coloured bright blue, instead of the usual method of spreading the poison on bread, no cases of adventitious poisoning were reported. 1,597 houses, in plague foci, were temporarily rat-proofed and

pulicidally disinfected, bedding etc. being passed through the steam disinfector. This temporary rat-proofing included the plastering up of rat holes, together with the bricking up and wire-netting of places permitting the ingress of the rodents into the houses. At the same time the furniture of the house was removed to permit of a thorough examination for rat holes and runs.

As a result of the extensive rat-proofing operations in the last two years, some 5,265 houses being done, a gratifying reduction of plague-infected rats has resulted: during the last quarter of 1910 there were 126 plague-infected rats found while during the same quarter of 1912 only four were found.

G. C. L.

**STRONG (W. M.).** *Annual Report, Medical Officer, Department of Native Affairs and Control, 1911-12.—Papua. Report for the Year ended 30th June, 1912.* pp. 160-162. [Printed and published for the Govt. of the Commonwealth of Australia. (1912. No. 87.).]

Strong states that in Papua, malarial infections are very common. In some villages nearly every native child shows signs of the disease. Although the type of parasite found is generally the malignant one, yet the infection in the case of natives seldom gives rise to any very serious condition of ill-health. The adult native of the coastal villages is usually more or less immune, and does not suffer appreciably from the disease as long as he remains in his own village, but if he leaves there he is liable to become infected. Coast natives are also very liable to become ill with malaria when taken into the relatively colder hill areas. Malaria is the cause of a certain amount of sickness on the plantations: it is unknown among the natives of the mountains: but, after visiting the coast these are very liable to become affected with the disease on their return home.

Leprosy has at times been reported from places on the south coast of the Territory. Mention is made of it in some of the old annual reports. In quite the early days of the country it is stated that a Polynesian immigrant became ill and died from a disease which was supposed to be leprosy. The case was never seen by a medical man, but he was seen by a layman who was familiar with the disease. Recently several cases have been found in the Mekeo villages and another small focus in the Trobriand Islands. The disease has, in all probability, been introduced into the country.

A comparatively little known disease is not uncommon on the south coast of the Territory. It is variously called rhinopharyngitis mutilans or gangosa. It might at first sight be mistaken for leprosy, and it is possible that some of the earlier reports about the latter disease may really refer to it. The exact nature of gangosa can hardly be regarded as settled. From what the author has seen of it he is satisfied that it is neither a form of syphilis, leprosy, nor lupus. It may perhaps be the disputed

tertiary or late stages of yaws, or perhaps it is a specific entity. The same disease is found in the Philippines and neighbouring islands, and has been described in other parts of the world; its comparative rarity in parts of the Territory other than the south coast suggest that it has been introduced.

Yaws is very common on the south coast of the Territory, but comparatively rarer in other parts. It is by no means improbable that it was introduced into the country when contact first took place with the outside world, and it has now spread to some of the outlying islands.

G. C. L.

#### BOOK REVIEW.

BESSON (A.). **Practical Bacteriology, Microbiology and Serum Therapy (Medical and Veterinary). A Text Book for Laboratory Use.** [Translation from the 5th French edition by H. J. HUTCHENS.] xxx. + 892 pp. With 416 illustrations. 1913. London: Longmans, Green & Co. [36s. net.]

The author in his preface to the 5th edition of his work states that recent advances in microbiology have necessitated an entire revision of the text. Most of the chapters have therefore had to be recast and much new matter has had to be incorporated. The original form of the book however is retained. All discussion upon matters of theory has been omitted with the exception of a chapter on Immunity and the Properties of Immune Serum. In the second part of the work notable changes will be found in the chapters on dysentery and meningitis while many modifications and additions have been introduced into the description and classification of the parasitic protozoa, especially the Piroplasmata, Leishmania, and Trypanosomata.

The edition, as in former ones, is clear, concise and thoroughly up to date. It is not necessary here to give a detailed list of the subjects dealt with in the work. It is sufficient to say that it covers everything required in an up to date laboratory and can be thoroughly recommended.

A word of praise may be added to Dr. Hutchens for his excellent translation. He has made certain necessary alterations at places and has added notes and additions, but these are clearly indicated either as footnotes or by being enclosed in square brackets. By this device the reader can see at a glance who is responsible for the statement.

Dr. Hutchens has also carefully revised the illustrations and has added many new ones.

These enhance the value of the work.

G. C. L.

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## TROPICAL DISEASES BUREAU.

TROPICAL DISEASES  
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[No. 7.]

## MALARIA.

HODGSON (E. C.). Report on the Work of the Central Malaria Bureau, 1911-12.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 51-52. (1913. Simla: Govt. Central Branch Press.)

The work of the Bureau has resulted in securing a complete set of all known Indian Anopheles and has aimed at getting together as many duplicate sets as possible for teaching purposes. Larva-eating fish have been secured and identified and new specimens have been added to the collection at the Bureau. A list of these is given. In addition specimens of duckweed of four species have been collected. These by growing in a thick layer on the surface of pools are said to hinder the growth of larvae. [In Algeria experiments in this direction with *Azolla* (Salviniaceae) did not succeed.]

Andrew Balfour.

BENTLEY (C. A.). i. A New Conception regarding Malaria.  
ii. Some Problems presented by Malaria in Bengal.—*Ibid.*  
pp. 61-70 and 71-84.

i. An epidemiological paper which advances a plea for a broader outlook, not only in the case of malaria but in all parasitic diseases. The author insists on the recognition of the difference between malarial infection and malarial disease. The former, *i.e.*, mere infestation by malarial parasites, is, he says, often as natural amongst primitive races as flea infestation is to a dog. He holds the theory that the occurrence of *epidemic* disease is always the result of a serious change in environment converting favourable into unfavourable conditions. Such changes may be due to the introduction of an unfamiliar parasite, the influence of unusual seasons, or the result of the great disturbances produced among primitive parasite-harbouring races by advancing civilisation. In this connection he cites the history of England and of Holland, both as regards malaria and other diseases. So far as malaria is concerned the paramount influence



in these countries was the improvement in agriculture, and this is what is chiefly required for dealing with rural malaria in India.

To this end the cultivating classes of the population should be educated, their standard of comfort improved and attempts should be made to induce capitalists to undertake agricultural operations on a large scale. The author thinks the function of the State lies mainly in providing for the education of the population, as though it may initiate reforms, the people must carry these into active practice and without their intelligent co-operation no scheme of improved sanitation can be made effective.

He believes that eventually civilisation and its attendant blessings will banish malaria from India as certainly as it freed England from this scourge of former days.

ii. There are areas in Bengal which have been largely depopulated and where malaria is common and severe. Bentley finds that the prevalence of fever is not due, as has been thought, to water-logging of the soil but is the result of a change in the relation of the Ganges to the land which owes to that river its origin. Hence it is in the older portions of Bengal where there are dead or dying rivers that the depopulated villages are found. The most common type of malaria is benign tertian, but malignant tertian is also frequently encountered. Quartan fevers appear to be rare. The commonest malaria-carrying anopheles is *N. fuliginosus* and its larvae are found in the waters of moribund rivers, in neglected tanks, pools, borrow-pits and other water collections where weed is present. The primary cause of the depopulation is not malaria but a decay of prosperity following upon impoverishment of the soil, which no longer receives the rich silt that it used to derive from the overflow of the subsidiary channels of the Ganges. The exhaustive planting of jute has also led to soil exhaustion. Sugar-cane acts like jute but rice impoverishes the land on which it is grown but slowly.

Poverty has followed the diminution in prosperity and as a result the malarial incidence has increased. According to Bentley the remedy is to be found in improved methods of agriculture fostered by the spread of education, the extension of co-operative credit banks, and the organisation of practical demonstrations of improved methods. Engineering precautions may prevent other threatened areas sharing in the disasters which have overtaken certain districts.

A. B.

FRY (A. B.). *Malaria in Bengal.*—*Ibid.* pp. 9-10.

Malaria in the Ganges Delta is endemic but not universal. It is confined to those parts which suffered from a severe epidemic in the middle of the 19th century. Parts which then escaped are now free from endemic malaria though their general condition shows no change.

Village sanitation and adequate treatment of the sick in order to reduce gamete carriers are the measures recommended. A proper system of sanitation depends largely on education of the natives, which is urgently required.

A. B.

HODGSON (E. C.). **Malarial Survey of Imperial Delhi.**—*Ibid.* p. 11.

An account of the local conditions favouring malaria at the site of the new city. There appears to be a close relationship between the spleen-rate, depth of subsoil water and percentage of dangerous anophelines present. The malaria rate showed a very definite rise in April and May though these are not rainy months. The author thinks that this may indicate that zygotes mature only in a definite temperature and is of opinion that special attention should be paid to humidity and temperature.

[This is interesting in the light of similar recommendations as regards the life-cycle of trypanosomes in tsetse flies.]

A. B.

GRAHAM (J. D.). **Notes on Anopheline Distribution in the United Provinces.**—*Ibid.* pp. 85-88.

Fourteen different species of Anopheles have been found in the United Provinces and a tabular statement of their distribution in the different and widely varying regions of this portion of Indian is the main feature of a paper which is chiefly of local interest.

A. B.

STOKES (T. G. N.). **Results of the Malaria Investigation in the Central Provinces.**—*Ibid.* pp. 59-60.

The question of endemic and epidemic malaria in the Central Provinces is briefly discussed. As regards the latter there is a note to the effect that scarcity of rainfall and high subsoil water are not essential to epidemic outbreaks and that the latter are not dependent on malarial endemicity. Two fatal cases of black-water fever in Europeans are recorded, one of which, however, may have been a case of malignant malaria.

A. B.

ADIE (Lt.-Col. & Mrs.). **Note of an Inquiry into Malaria and Mosquitoes in the Kashmir Valley.**—*Ibid.* pp. 91-94.

The result of this enquiry was to show that in Srinagar and its neighbourhood there is no endemic malaria; there are practically no anophelines. A certain amount of imported malaria is found but the risk of spread is very slight.

A. B.

PERRY (E. L.). **Malaria in the Jeypore Agency Estate.**—*Ibid.* pp. 11-13.

Major Perry thinks there is evidence to show that in the Jeypore region the stream-breeding species of anopheles, *i.e.*, *listoni*, *theobaldi*, *jeyporiensis* and *maculipalpis* are largely concerned in the transmission of quartan malaria and that the great rarity of malignant tertian, save where *culicifacies* is present, points to this mosquito being the chief vector of *P. falciparum*. [See paper by KNAB reviewed in this number.] Perry believes the Jeypore estate could be greatly benefited by careful jungle-clearance operations.

A. B.

HORNE (J. H.). *Malaria in the Madras Presidency. Notes on the Statistics of the Past Ten Years.*—*Ibid.* pp. 53-57.

As a result of his enquiry the author concludes that:—

1. "Only one district shows evidence of epidemic malaria: its soil is barren, its climate dry, its rainfall scanty but, when heavy, liable to cause floods. The epidemics were localised and associated with increased rainfall but not with drought in the previous year. The epidemics were followed by a marked reduction in the birth rate of the subsequent year.

2. "Normally, deaths are most frequent at the beginning and end of the year. This rise is accentuated in districts known to be malarious.

3. "Areas known to harbour endemic malaria appear to be associated with a high infantile mortality, but may possess a high birth rate."

A. B.

RIEUX (J.) & HORNUS (P.). *Notes sur le Paludisme dans le Maroc Occidental.*—*Arch. de Méd. et de Pharmacie Militaires.* 1913. July. Vol. 62. No. 7. pp. 1-31.

Foci of malaria are common in Western Morocco. Amongst the French troops stationed there the disease was specially prevalent in August and September 1911, and September and October 1912, but throughout the latter year it was much less in evidence than during 1911. The type was benign and the mortality low, thanks partly to this and partly to the efficiency of quinine. In every case the diagnosis was confirmed by microscopical examination. The quartan parasite was never found, benign tertian or double tertian and aestivo-autumnal being the types in evidence. Mixed or associated forms were occasionally seen. Charts are given showing the varieties of fever encountered, and the tendency of the malignant forms to lose their intermittency and become continued is duly noted. The authors accept the view that each of the three forms of the malarial parasite represents a distinct and definite species and advance arguments in favour of this view as regards the two kinds found in Morocco. The paper ends with notes on spontaneous cure and the use of quinine both as a prophylactic and as a curative agent.

A. B.

v. d. HELLEN. *Notizen über Malaria bei Eingeborenen in Togo (Westafrika).* [Notes on Malaria amongst the Natives in Togo (West Africa).]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 13. p. 461.

A native assistant who accompanied the author on a journey through a malarious district for a period of over a year did not suffer from malaria until about nine months later. During these later months he resided continuously in a comparatively mosquito-free centre and did not take quinine. In this short time therefore he had lost his relative immunity. He admitted having been bitten by mosquitos while on a two days' furlough to his village and the malaria developed two or three weeks thereafter. The natives in Togo only suffer from malaria in childhood which, however, does not seem to influence the infant mortality. The latter is due to errors in nutrition. The author argues that his

demonstration of the loss of relative immunity in a native indicates that it would be unwise to carry out extensive preventive measures in a region where the possibility of the adult natives acquiring infection is only slight.

A. B.

**KNAB (Frederick).** *The Species of Anopheles that transmits Human Malaria.*—*Amer. Jl. Trop. Diseases & Preventive Med.* 1913. July. Vol. 1. No. 1. pp. 33-43.

One of several papers forming the first number of a new journal which appears under the auspices of the American Society of Tropical Medicine. It contains no new work and is intended to indicate certain lines of research which have been neglected in the past. After criticising the new systematic nomenclature of the old genus *Anopheles*, the author, confining himself to the American *Anopheles*, tabulates the valid species with their correct names. Out of the thirty-four mentioned the following eight have been definitely proved to be malaria carriers:

<i>albimanus.</i>	<i>quadrimaculatus.</i>
<i>argyritarsis.</i>	<i>pseudomaculipes.</i>
<i>crucians.</i>	<i>pseudopunctipennis.</i>
<i>intermedium.</i>	<i>tarsimaculata.</i>

Attention is drawn to the fact that, in all probability, the same species of mosquito may behave differently towards the different species of malarial parasites, and work both in New Orleans and Formosa is cited in favour of this view. Thus *A. crucians* acts as the transmitter of aestivo-autumnal fever alone while *A. quadrimaculatus* does not convey this form but is efficient with benign tertian and quartan malaria.

An interesting subject requiring further investigation is the relation of the habits of different species of *Anopheles* to malaria transmission. The frequency of blood meals increases the chances for acquiring the parasites. Hence the more voracious the mosquito the more likely is it to be a carrier of infection. Again 'domestic' *Anopheles* are more likely to be incriminated than those classed as 'wild.' Other phases of the study of *Anopheles* requiring attention are the relative longevity of different species and their local distribution as governed by topographic conditions. The author advances a plea for the prosecution of these and other investigations required for the proper control of the mosquito.

A. B.

**LALOR (N. P. O'Gorman).** *Note upon some Unusual Forms of the Parasite of Pernicious Malaria, found at an Endemic Blackwater Fever Centre, in Blood Smears from Certain Children.*—*Indian Med. Gaz.* 1913. July. Vol. 48. No. 7. pp. 253-254.

At Wuntho in Upper Burma in the month of May the author found sporozoites in the salivary glands of a single *A. listoni* var. *albo-apicalis*. These, he thinks, were peculiar in the colouration of their cytoplasm by Leishman's stain and the arrangement of their chromatin. It is possible they may have been derived from a peculiar form of parasite which he discovered in the blood of

children in February and the earliest form of which he describes as a spore surrounded by a blue-staining envelope. These envelopes are said to rupture and the freed spores to become incorporated with the nuclei of certain large mononuclear cells, apparently of endothelial origin. Within these the spores grow. Finally each separate spore undergoes schizogony and, from each, six large spores result each of which possesses an envelope derived from the altered nucleus of the host cell. Finally these spores become free and either invade red cells, appearing as ordinary malignant plasmodia, or change into malarial crescents, which differ slightly from the ordinary gametes of *P. falciparum*. Invaded red cells are profoundly altered and the intra-corpuseular parasites have a coarser cytoplasm and are more pigmented than is usual with the ordinary parasite of malignant fever. Lalor believes them to be a different and distinct type. If so, its periodicity has yet to be determined. A coloured plate shows the sporozoites, the spores, the intra-nuclear forms(?), those found in the red cells, and the crescents.

A. B.

## CLINICAL.

BATES (John Pelham). *A Review of a Clinical Study of Malarial Fever in Panama.*—*Jl. Trop. Med. & Hyg.* 1913. July 15. Vol. 16. No. 14. pp. 209-213.

This is a third instalment of the paper already dealt with in this *Bulletin* (Vol. 2, p. 150 and p. 221) and is concerned with malarial anaemia. The author mentions the two factors generally regarded as being operative in producing the anaemia, i.e., the destruction of the red cells by the growth of malarial parasites within them, and the action of a haemolysin of unknown nature. He discards the theory of intravascular migration of parasites advanced by ROWLEY-LAWSON\* to explain the supposed inequality in the number of parasites present in the peripheral blood and the amount of anaemia following acute malarial attacks. After citing the work of various observers and especially that of DIONISI, both as regards the features of malarial anaemia and the ultimate increase which occurs in the number of red cells despite repeated febrile attacks, he gives an account of his own work. It began in 1910. Confined at first to secondary anaemias of long duration it later embraced the acute secondary anaemias following repeated attacks of primary malarial infection. The total number of cases under observation is not stated but the author groups them as follows:

1. Acute malaria cases with irregular attacks extending over a period of from two to three months—outside patients.

2. Hospital patients with a history of from five to thirteen attacks of fever extending over a period of from three to six years.

3. Cases of chronic malaria in natives who had lived amid continuous malarial infections and suffered repeatedly from fever.

In every case the number of red cells was ascertained and also the haemoglobin value either by Dare's or Sahli's method. The

\* *Arch. Internal Med.* 1912. Apr. 15. Vol. 9. No. 4. pp. 420-444.

patients in the first two groups were under rather favourable general conditions, could obtain good food and were promptly treated when attacked by fever. In giving details of cases the author does not distinguish those of the first from those of the second group, but as a result of his observations concludes that where the anaemia is solely dependent on malarial infection one may expect first a sudden and marked loss both of haemoglobin and red cells. At a certain stage the losses cease and, if the attack is cured spontaneously or by treatment, a rapid rise both in red cells and haemoglobin occurs in the afebrile interval. The next attack produces a lessened effect and so on until finally the losses cease altogether. At this stage of tolerance there is a reproduction of red cells and haemoglobin in excess of the losses, that of the corpuscles usually exceeding that of the haemoglobin.

In chronic malaria on the other hand there is no tendency to improvement in the apyrexial intervals but either a stationary or progressive anaemia. Bates seeks to show that this anaemia is not due to malaria but to a concomitant uncinarial infection, or to this plus semi-starvation as a result either of lack of food or of a dietary poor in protein value and containing much coarse fibrous material. Cases with very low haemoglobin values, *i.e.*, 13 to 21 per cent. and red cell counts of from 850,000 to 1,703,000 per cubic millimetre were attributed to ankylostome infection while those with higher haemoglobin estimates seemed in the main to be due to poor food. Exhaustion of the haematopoietic organs is shown not to be operative, and the author thinks that MARCHIAFAVA and BIGNAMI who advanced this hypothesis to explain post-malarial anaemia were dealing with cases complicated by ankylostomiasis, which is common in Italy. He is of opinion that the term malarial cachexia is misleading, serves to misdirect efforts at treatment, and ought to be abandoned in medical nomenclature. [It will be noted that Bates, beyond speaking of an unknown haemolysin, does not consider the part played by haematin in producing anaemia, a subject dealt with by BROWN in a paper reviewed elsewhere in this number.]

A. B.

MARCHIAFAVA (E.). Ueber *Malaria perniciosa*.—*Deut. Med. Wochenschr.* 1913. Aug. 14. Vol. 39. No. 33. pp. 1577-1581.

After a reference to his earlier work along with BIGNAMI the author mentions the occurrence, at the present time, of cases of pernicious malaria in the Roman Campagna. These tend to terminate fatally despite quinine treatment. He gives an account of several such cases and recalls the description of the specific parasite given by CELLI and himself in 1889, together with its effect on the red cells and the changes produced by its action in the internal organs. He has found it necessary to recur to the subject because recently certain authors, *e.g.*, DEADRICK, have stated that both quartan and simple tertian infections may be pernicious in type. While these forms of malaria can present severe symptoms, such as stupor, depression, prostration, &c., more especially in children and old persons, and may, in the

former, be associated with convulsions, yet these symptoms vanish quickly at the end of the febrile attack and they do not tend to a fatal issue even when treatment is delayed. Two such severe cases are cited, the one a simple, the other a double benign tertian and mention is also made of a fatal case of haemoglobinuria following quinine administration in a case of simple tertian fever. In this case there was the most severe renal inflammation the author has ever seen. At the same time he holds that pernicious malaria is a distinct entity and he lays special stress on the enlargement of the spleen in this type of malaria. The organ is not very large, its capsule is smooth, thin and transparent, and shows no evidence of a previous increase in thickness. The pulp is very soft and sometimes flows out, as it were, when the capsule is incised. The splenomegaly gives the impression of being recent and not an acute exacerbation occurring in the course of a chronic inflammation. The condition is like that found in typhoid fever save that the malarial spleen is pigmented and its pulp is softer. It is thus evident that pernicious malaria is always a recent primary infection. At the most there is only one relapse. The author thinks that a long standing malarial infection confers a certain immunity against severe malaria in comparison with what is found in those who are attacked without having suffered from a previous infection. In these latter the parasites multiply with great rapidity and are sometimes present in enormous numbers especially in the internal organs and bone-marrow. An account is given of a case in which the brain capillaries exhibited varicose extensions so crowded with parasites containing red cells that the condition was diagnosed as a true, globular stasis.

The author goes on to speak of quinine resistant strains of the parasite and of the danger of giving quinine at certain stages, as the drug may produce a precipitate sporulation leading to death as a result of injury to the nerve centres and to poisoning from the toxins set free. Sometimes when quinine has been freely given and the parasites have disappeared from the peripheral circulation death may ensue and very few parasites be found post-mortem except crescent forms, and these chiefly in the bone-marrow. In such cases there are often punctiform haemorrhages in the brain substance. When speaking of preventive measures the author advocates the establishment of hygienic stations in the malarial districts of Italy for the instruction of the agricultural labourers, &c., where quinine prophylaxis and that *prompt* treatment which is often so essential in pernicious malaria can be carried into effect. He also mentions the value of JAMES's modification of Ross's thick film method,\* though he is careful to point

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\* Spread a fairly large drop of blood in the form of a circle about three-quarters of an inch in diameter. Place the slide on a flat surface and allow the blood to dry. Immerse in a mixture of ten drops of commercial HCl in 50 cc. of ethyl alcohol until the haemoglobin has been dissolved out. Wash very thoroughly in running tap water for ten to twenty minutes. Dry either in the air or by blotting. Stain with Romanowsky undiluted stain for two or three minutes, then dilute freely, carrying out this dilution in successive stages, the whole process occupying about ten minutes. Finally wash in tap water until no more blue colour comes from the film. For further details consult original paper in *Southern Medical Journal*. Vol. 4. 1911. p. 698. The above summary, however, gives the essentials of the method.—A. B.

out that it is not suitable for those who are tyros in the examination of blood films for the diagnosis of malaria.

A. B.

ACTON (H. W.) & KNOWLES (R.). *Latent Malaria*.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras. Nov. 18, 19, 20, 1912. pp. 22-23. (1913. Simla: Govt. Central Branch Press.)*

This is an account of work undertaken to give greater precision to the present method of diagnosing latent malaria. The first part deals with the definition of latency and sub-divides latency into distinct phases, such as the latency of the incubation period, that preceding recrudescence and that preceding relapses, these latter two being differentiated. The authors believe in parthenogenesis as one of the factors concerned in relapse. Two indirect tests for latent malaria are employed:—(a) The urinary urobilin test; (b) The leucocyte count.

The following summary sufficiently explains their views:—

1. "The presence of urobilin in the urine in large quantities indicates that haemoglobin is being destroyed. The blood destruction, except in certain well defined diseases, is due to destruction of erythrocytes by malarial parasites. If there is no fever present the patient is in the latent stage of the disease.
2. "In the latent period of relapse the asexual cycle is absent in the peripheral blood and gamete formation is occurring. The urobilin consequently is diminished in amount, unless the functions of the liver are impaired in which case urobilin may be increased.
3. "The absence of urobilin from the urine indicates either that blood destruction is not occurring or that the liver is still able to deal with the products of haemoglobin destruction. In such cases other tests for latent malaria must be applied.
4. "A leucopenia of 2,000 to 7,000 per c.mm., or a leucocytosis, 16,000 or more, in the absence of other causes, is suggestive of latent malaria.
5. "A high relative mononuclear percentage is also suggestive that protozoal parasites are present.
6. "A fluctuating leucocyte count with a high mononuclear percentage at the leucopenic stage is very suggestive that malarial parasites are still present somewhere in the body.
7. "The increase in the total mononuclear and hyaline cells is not a real but an apparent one, due to fluctuations in the number of the polymorphonuclear leucocytes."

Tables and charts are mentioned but have not been reproduced.

A. B.

HENSON (Graham E.). *The Diagnosis of Malaria*.—*Southern Med. J.* 1913. July 1. Vol. 6. No. 7. pp. 423-426.

A plea for laboratory diagnosis in suspected malaria and a suggestion that an effort should always be made to take blood smears for diagnosis at a time when it is probable that the parasites are in greatest profusion in the peripheral blood. In benign tertian infections this is from four to six hours after the chill and thereafter to within six hours of anticipated sporulation. In quartan the parasite is more easy of detection throughout the entire cycle and hence the timing of a smear is not so important. In aestivo-autumnal types two hours after the chill or exacerbation and thereafter for two or three hours will be found the most



likely periods. When, however, crescents have formed they may be found at any time. In a discussion which followed the reading of the paper BASS of New Orleans stated that there never occurs a time when there are not enough parasites in the blood of a patient who is having chills and fever from malaria but that the parasites can be found. The proper time to take blood therefore is whenever one sees the patient for diagnosis. [While the latter part of the statement is no doubt true, Henson's recommendations are likely to be helpful especially to those with little experience in blood examination or little leisure to conduct it in a searching manner]. A diagnosis is apt to be specially difficult if quinine has been administered. In such a case a tentative diagnosis of malaria may be made when:—

- (a) A clinical history of malaria is obtainable.
- (b) The patient has an enlarged spleen.
- (c) A leucopenia is present.
- (d) The red cell count is below normal.
- (e) The haemoglobin is reduced.
- (f) A differential count shows an increase of large mononuclears.

A. B.

FREEMAN (James V.). *The Incidence of Malaria in the Puerperium.*—*Southern Med. Jl.* 1913. July 1. Vol. 6. No. 7. pp. 429-430.

The author insists on the importance of distinguishing between malaria and sepsis in the puerperium. Malarial infection in childbed produces a fever within the first week without definite chill. Unless the patient is known to be a malarial subject, it can only be diagnosed by blood examination. This, together with absence of the usual signs of sepsis, febrile periodicity and the application of the therapeutic tests, enable a diagnosis of malaria to be established. As was pointed out in a discussion on the paper the mere presence of malarial parasites in the peripheral blood does not always justify one in assuming that malaria is the sole cause or indeed even a cause of the febrile condition.

A. B.

KAHN (Ida). *Some Experiences with the Subtertian Fever in Kiangsi.*—*China Med. Jl.* 1913. July. Vol. 27. No. 4. pp. 231-236.

In Kiangsi subtertian malaria causes a greater mortality than tuberculosis and no age is exempt from its attacks, which are sometimes of lightning-like rapidity. The bilious form is very common and a severe case successfully treated by intramuscular injections of quinine bi-hydrochloride and stimulants is recorded. The algide type is not so frequent but a form of malarial bronchopneumonia in children is far from rare. If sufficient quinine is not given, the lung symptoms will not quickly subside and yet if the quinine is pushed there is danger of amblyopia, as in a

case described by the author. Here, however, the quinine was continued, but given at longer intervals, and the sight was rapidly recovered and later appeared to be quite normal.

A. B.

WOOLLEY (J. M.). **Malaria in the Andamans: Fever with Jaundice Cases.**—*Indian Med. Gaz.* 1913. July. Vol. 48. No. 7. pp. 266-267.

This is an account of a peculiar "Fever with Jaundice" which has been noticed in the Andaman Islands during the past three or four years. Hitherto it has been considered malarial but the writer thinks it may yet have to be classed as a distinct entity. The jaundice appears on the third or fourth day of the illness and is often intense. In bad cases there are petechial haemorrhages in the skin and bleedings from the gums, delirium and a feeble and rapid pulse. Forty per cent. of the cases die, usually from cardiac syncope. The post mortem appearances are summarised but are not specially characteristic.

Malarial parasites are not found in the blood but in fresh blood-films small rod-like, or round, non-pigmented, motile bodies, have been seen. They cannot be stained but the author thinks they are parasites and cause the disease. [They are probably merely artefacts, the so-called "Maraglianos"]. The treatment of this fever is entirely symptomatic. Quinine is of no avail.

A. B.

#### TREATMENT.

- MACGILCHRIST (A. C.). i. **Pharmacological Action and Uses of Quinine.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 17-18. (1913. Simla: Govt. Central Branch Press.)
- ii. **Intravenous Injections and Subcutaneous Infusions of Quinine Salt Solution.**—*Ibid.* p. 97.

i. The author believes in large doses of quinine if treatment is to be efficient. Such doses taken with or soon after meals at eight-hourly intervals keep the concentration of quinine in the blood at a high point throughout the twenty-four hours. There are no objections to giving quinine during the fever period. As regards prophylaxis it is stated that fifteen grains of quinine taken on two consecutive days each week keep the malarial parasites in subjection as long as such treatment is in force, but it does not disinfect the patients. Relapses may occur when the prophylactic treatment is stopped but such treatment does not tend to increase the liability to, or the occurrence of relapses after its cessation. Little definite is known regarding the action of quinine on the blood vessels, but its action on the uterus under different conditions has been noted. It will greatly increase the expulsive contractions of the already active pregnant uterus but cannot induce or originate expulsive contractions in the quiescent pregnant uterus. The action of quinine as a local anaesthetic practically amounts to a chemical resection of the nerves. The author concludes with a plea for a more extended

use of quinine base in a pure precipitated amorphous form. Its advantages are:—

1. Sparing solubility.
2. Comparative tastelessness.
3. Absorption as quick as with a salt.
4. Bulk and weight small.
5. Non-haemolytic action.
6. Cheapness.

Quinine base can be administered intravenously in two ways:—

- (a) As a 1 in 135 solution in 33 per cent. alcohol in saline.
- (b) As a 1 in 2,000 or 5,000 solution in saline.

ii. The author states that, although in a previous paper\* he gave 1 in 150 as the strongest solution of a quinine salt which could safely be introduced intravenously without danger of thrombosis, he did not recommend the use of solutions of this concentration as they are very irritating. As he mentioned, seven grains or so of quinine bi-hydrochloride should be dissolved in two or three pints of normal saline. He refers to the new method of JAMES, *i.e.*, subcutaneous infusions [see this *Bulletin*, Vol. 2, pp. 151-152], and thinks it must cause extensive local disintegration of the tissues at the seat of the infusions, owing to the concentration of the solution used.

[JAMES's method has been used in several cases in the Anglo-Egyptian Sudan with excellent results and no untoward effects.]

A. B.

JUSTI (Karl). *Zur Methodik der Chinindarreichung bei Malaria.*

[On the Methods of Quinine Administration in Malaria.]—

*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Aug. Vol. 17. No. 15. pp. 505-522.

This paper is largely a compilation and criticism of the views of various authors as to the best way of administering quinine in malaria. The author enters very fully into the matter and, in discussing the oral methods as compared with injections, considers the pharmacological and clinical data on which a conclusion as regards their respective merits must be based. He is apparently unacquainted with some of the recent Indian work on the subject and confines himself in the main to that of German observers. At the same time he has had an extensive experience at Hongkong, extending over 25 years, and he carefully explains the reasons which have led him to adopt the procedure he now follows and which he recounts in detail.

He agrees with WERNER of Hamburg that, in oral administration, NOCHT's method of fractional doses, *i.e.*, 0.2 grammes every two hours from 7 a.m. to 3 p.m. daily is better than a single, large dose, *i.e.*, one gramme, as it causes less vomiting, and the sickness, if it occurs, is less severe. He finds that WERNER's intravenous injection of 1½ grammes of quinine urethane in 200 c.c. of physiological salt solution, as employed

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\* *Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India (New Series).* No. 41. 1911.

in severe comatose cases, is better suited for hospital than for private practice as it presents technical difficulties and must infallibly lead to delay if used where the necessary apparatus cannot always be kept ready at hand. He does not agree with WERNER's conclusions as to the comparative inefficacy of intramuscular injections and believes that WERNER's views on this method were based on a misconception of the action of quinine, so administered, on the malarial parasites.

As he says, the objections to the ordinary injection methods, subcutaneous and intramuscular, are that the quinine is supposed to be less active than when given by the mouth and that there are apt to be unpleasant sequelae in the way of pain, local necrosis and suppuration. He discusses these objections in the light of what is known regarding the pharmacological action of the drug, admitting that we are still in the dark as to some of its effects and the changes it undergoes in the body. Various authors are quoted to show that both in subcutaneous and intramuscular injection this destruction and retention of quinine in the tissues, &c., is as great as when it is given *per os*. If, as MARIANI holds, and he is supported by GIEMSA and SCHAUHMANN, the amount of quinine excreted by the kidneys is proportional to its anti-parasitic power, then we must conclude that the injection method has only half the value of the oral. ZIEMANN, however, is of opinion that it is not the eliminated quinine but that used up by the body, *i.e.*, united to cell protoplasm, which plays the chief part in the therapeutic action. That the matter is not so simple is indicated by the recent researches of PLEHN and GROSSER who have shown the special rôle played by the endothelial cells of the vessel walls in rapidly removing quinine from the circulating blood. A similar selective action on the drug is in all probability exerted by the malarial parasites themselves. PLEHN believes their destruction takes place in the circulating blood and not in the internal organs (GIEMSA and SCHAUHMANN).

After a further consideration of the experimental work, both of the authors already mentioned and that of ARNAUD, Justi decides that the data available do not support the objection which has been raised on pharmacological grounds to the employment of the injection methods. He then passes to a consideration of the objection advanced on clinical grounds and is here able to speak with considerable authority as he has had experience of nearly 2,000 injections. In only three cases did abscesses result and these, for reasons stated by the author, cannot fairly be considered as failures. In dealing with the question of tissue destruction he gives the details of his aseptic technique on which so much depends. Here there is nothing special to note but he lays great stress on the choice of the place where the injection should be made. This, according to his view, is the gluteal region and only the gluteal region, both in the case of male and female patients. The neighbourhood of the sciatic nerve is to be avoided and he is strongly opposed to mere subcutaneous injection. [Apparently he is not familiar with the modified method introduced by JAMES in Panama (see this *Bulletin*, Vol. 2, pp. 151-152).]

Provided the precautions mentioned are adopted there is no objection to a daily injection. Observations are recorded showing the efficacy of the injection method as regards its effects on the parasites (benign tertian and malignant), on all three types of fevers, and on the enlarged spleen both in acute and chronic cases of malaria. For details the original paper must be consulted but it may be said that the author finds that, as far as the disappearances of ring forms and older schizonts is concerned, the intramuscular injection is just as rapid in its effect as the oral administration while, possibly, its action is superior in the case of gametes. In a brief summary he says that subcutaneous injections are not to be recommended, that intramuscular administration is quite as useful as oral administration and may be used in preference to NOCHT's methods in case of sickness and vomiting, and even in those exhibiting somnolence and coma so long as intravenous injection is not imperatively indicated.

In his own practice the author begins with one injection repeated every 24 hours and continued for some time after all parasites have disappeared. In pernicious cases never less than six injections are given. He then exhibits quinine by the mouth, one gramme on each of two successive days, usually Saturday and Sunday, *i.e.*, about 30 grains in the course of a week. In suitable cases, however, he employs NOCHT's method from the beginning, reverting to the injection if sickness and vomiting supervene.

A. B.

WATERS (E. E.). *The Pure Amorphous Alkaloid (of Cinchona)*. [Correspondence.]—*Indian Med. Gaz.* 1913. July. Vol. 48. No. 7. p. 285. *The Amorphous Alkaloid of Cinchona*. [Editorial.]—*Ibid.* pp. 273-274.

Major Waters writes to explain the nature of the pure amorphous alkaloid of cinchona previously described by him [see this *Bulletin*, Vol. 1, p. 650]. It is a sticky, treacle-like substance which, owing to its peculiar physical characteristics, is difficult to incorporate in large doses in tablet form. So far one and two grain tablets only have been issued. Confusion has arisen because in addition to this *pure amorphous alkaloid* there is the amorphous cinchona alkaloid. This is only 40 per cent. of the strength of the pure alkaloid, from which it differs physically, and hence can be issued in 3 1/3 grain tablets. To prevent confusion Waters suggests the name "Laverain" (after LAVERAN) for the pure amorphous alkaloid, which is a very complex body. The Editor of the *Indian Medical Gazette* points out that as Quinoidine is the old name for this drug it must remain in force.

As a result of Major Waters' letter the *Indian Medical Gazette* has an editorial on the subject which mentions both these alkaloids under the names of Quinoidine and Residual Alkaloid respectively. It points out that as the dose of the former is small, a three grain tablet containing a one grain dose, is quite satisfactory. The Government Quinologist furnishes a useful general note on "The Amorphous Alkaloid of Cinchona Bark" which should be consulted by all interested in the subject. Residual

alkaloid is really an amorphous mixture of *all* the alkaloids of cinchona bark, having an average composition of:—

Quinine	...	...	...	3
Cinchonidine	...	...	...	2
Quinidine	...	...	...	20
Cinchonine	...	...	...	35
Quinoidine	...	...	...	30
Water, Ash, &c.	...	..	...	10
				100

The effect of the mixture is not due to any one alkaloid alone, for all are effective in the cure of malaria while it is supposed that the amorphous state of the preparation renders it more readily assimilated.

Quinoidine in the form of its salts can be separated out owing to their greater solubility. Some traces of the other alkaloids remain in the product finally obtained, which is evidently a mixture of substances as it varies in consistency according to the kind of bark from which it has been derived. At present it is being prepared in India from Residual Alkaloid, but it may be necessary to select and grow strains of cinchonas giving a larger yield of this particular alkaloid.

A. B.

Muir (E.). *The Diagnosis and Treatment of Chronic Malaria and Kala-azar.*—*Indian Med. Gaz.* 1913. July. Vol. 48. No. 7. pp. 267-268.

The author has extended his kala azar treatment (see *Kala Azar Bulletin*, 1912, July, No. 3, 125-126) to cases of chronic malaria but he has modified it so that, instead of giving hypodermic or intra-muscular injections of a very strong acid solution of quinine sulphate, he now relies on the following mixture to produce the local irritation which he believes confers the benefit:—

	Drachms.			
Turpentine	...	...	...	1
Camphor	...	...	...	1
Creosote	...	...	...	1
Sterilised olive oil	...	...	...	2½

The camphor and creosote are mixed first and then the turpentine and oil added. 5-15 minims are injected into the muscles on both sides of the body, the latissimus dorsi or glutei being the most suitable sites. There is little pain at the time of the injection but pain and swelling come on later proportionate to the amount injected and to the susceptibility of the patient. In chronic malaria the reaction is more marked than in kala azar. One or two injections in moderate cases and up to fifteen or twenty injections given weekly in more advanced cases restore the patient to a marvellous degree of health and strength.

A. B.

KOPYLOW (N. W.). Ueber Splenektomie bei Malaria-affektion der Milz. [Splenectomy in Malarial Affections of the Spleen.]—*Arch. f. klinische Chirurgie*. 1913. June 17. Vol. 101. No. 3. pp. 708-734. With 6 text-figs.

This paper opens with an interesting historical account of the operation for removal of the spleen which, as far as chronic malaria is concerned, was first performed by KÜEHLER in 1855. The patient died, but shortly afterwards DORSAY operated successfully in a similar case. The author describes the changes induced in the spleen by chronic malaria and the functional disturbances produced by the enlarged and altered spleen in neighbouring organs. He also deals with the subject of "floating spleen," which is most common in women.

Splenectomy is indicated for malarial affections of the spleen—

"1. In splenic rupture and torsion of the pedicle, the latter tending to occur in floating spleen and causing symptoms of peritonitis. In these cases the operation appears to be vitally indicated and must be performed at once, otherwise death will undoubtedly occur.

"2. In cases of enlarged movable spleen—floating spleen. It is relatively easy to perform the operation here; nevertheless pedicular torsion with all its severe sequelae must always be anticipated.

"3. In cases of large, immovable, painful spleen, in which medical treatment has given no noticeable result."

Although in cases of slight hypertrophy of the liver, moderate cachexia and advanced age the mortality percentage is still fairly high (about 30 per cent.), the operation is justifiable; but it is contra-indicated

"1. When there is considerable cachexia with hyperaemia and marked diminution of the haemoglobin content (below 40 per cent.).

"2. When there is severe atrophic cirrhosis.

"3. When the general condition is bad with diseased digestive and uro-genital organs.

"4. When there are very extensive adhesions, especially to the stomach and diaphragm."

The author gives a careful account of 13 splenectomies performed in the Balachnin Hospital of the Naphtha works, close to Baku in the Caucasus. Ten of these were his own operation cases and nearly all the patients belonged to the peasant class.

Attacks of malaria were observed in three patients during their stay in hospital after operation. Other complications and sequelae are mentioned. There follows a valuable section dealing with the operative technique, considered under the following headings, (a) Section through the abdominal wall, (b) Ligature of the pedicle, (c) Separation of the adhesions, (d) Closure of the abdominal cavity.

Death after splenectomy is stated as being due to shock, haemorrhage, peritonitis, pneumonia, gangrene of the walls of the stomach and intestine, or as the result of malarial changes in the liver and spleen.

A mortality table compiled from various sources is given, from which it appears that the mortality ranges between 7 and 60 per cent. In Kopylow's own cases it was 23 per cent., or, excluding a case operated on *in extremis*, 16 per cent.

When successful the operation gives good results, the patients being able to resume their work. The author thinks there is a distinct future for splenectomy in malarial affections of the spleen, but he is careful to point out that the technique must be improved and care taken to follow correct indications for operative interference.

Five of the six figures illustrating this useful paper are photographs of spleens removed by the author.

A. B.

DEGORCE (A.). *Accès Palustres Mortels ou Tenaces observés après la Splénectomie.*—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. June. Vol. 4. No. 6. pp. 299-306.

An account of three cases showing that splenectomy may be followed by fatal or very severe attacks of malaria. In two of the cases death resulted and was undoubtedly due to the operation, which was not in any way complicated and did not occupy an unduly long time. There was no doubt that the surgical procedure was to blame and energetic treatment with quinine failed to save the patient. The third case was an example of a severe malarial attack occurring fifteen days after splenectomy and characterised by a fever very resistant to quinine given both by injection and by the mouth. The author discusses the cause of these accidents.

Traumatism might explain the first two cases where the attacks quickly followed the operation, but he is more inclined to believe, a belief strengthened by the history of the third case, that the extirpation of the spleen interfered with the defensive mechanism of the organism. He concludes, therefore, that splenectomy is only justifiable in those cases where the splenomegaly results in much suffering and where the least injury is apt to cause rupture of the spleen. When splenectomy is decided upon, even if no parasites can be found in the peripheral blood, it is advisable before operation to treat the patient thoroughly by a series of quinine injections.

A. B.

#### PROPHYLAXIS.

LEGENDRE (J.). *Prophylaxie du Paludisme en Italie.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 468-476.

To TERNI belongs the credit of being the first in Italy to suggest the utilisation of fish for the destruction of mosquito larvae. The fish he recommended were carp, tench and eels, which not only possess a hygienic value but are of economic importance and have thus an advantage over "millions" and other small species of fish which are only of use as destroyers of larvae. TERNI examined the stomach contents of small tench taken from the water in rice cultivations and found 60 to 80 larvae of anophelines and other mosquitos in each of them. The author



gives an account of the systematic pisciculture now conducted in the rice-growing areas of Lombardy. Carp are employed, being introduced as young fry which, thanks to abundant food, rapidly increase in size and weight. During the winter they are kept in a special basin and fed with kitchen scraps. In the summer they are reintroduced into the water of the rice fields. After their second season in the rice fields the carp at the age of 16 months are eaten or sold at a good profit. The rice culture benefits from their presence as less weeding (a costly operation) has to be done and it is found that in the carp-stocked cultivations the yield of rice is equal or even superior to that elsewhere. There is no doubt that from the mere economic standpoint the pisciculture is a great success and it is being adopted with enthusiasm throughout Lombardy, the species of carp employed being *Cyprinus carpio* var. *specularis*, which is obtained from Germany. Sufficient evidence has not yet been accumulated to permit a definite statement being made as regards the anti-malarial value of this measure but there is enough to show that it is really beneficial and likely to have a future. The author recommends the adoption of pisciculture in Madagascar and Indo-China.

The paper then considers other anti-malarial measures employed in Italy: quininisation, hydraulic works and mechanical protection. The so-called "quinine preventive" method of **CELLI**, the pre-epidemic cure of **GOLGI** and the plan followed by **TERNI** receive brief mention. The last mentioned consist of an intramuscular injection of the following formula:—

	Grammes.
Basic chlorhydrate of quinine ... ..	3
Ethylurethane ... ..	2
Oil of vaseline ... ..	5
Emulsify by heat.	

In heavily infected localities the injection is repeated once a month and this method is recommended for use in the Colonies and in countries where it is impossible to establish a regular daily quininisation.

A. B.

**CELLI (A.). Malaria in Italy during 1910.** [Translated from the Original by Major N. P. O'Gorman LALOR. With a Preface by the Translator.]—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 113-150. (1913. Simla Govt. Central Branch Press.)

The translator summarises the conclusions of Celli as set forth in this long report as follows:—

1. "That quinine taken at an approximate daily rate of three grains in the morning and three at night, throughout the fever season by a healthy individual in a severely stricken malarial locality, practically guarantees him against the risk of malarial infection.

2. "That quinine similarly consumed by every individual, whether presumably healthy or malarially suspect, in a malarial community, affords similar and proportional guarantee as regards the community.

3. "That quinine taken in the prophylactic quantity mentioned, is absolutely devoid of harmful effect, proximate or remote, to the consumer.

4. "That the risks to which subjects of chronic malarial infection are said to be exposed by daily quinine consumption, viz.:—

"(a) Intracorporeal development of haemamoebic types resistant to quinine (*chininfaste*n Malaria Parasiten) and

"(b) Quinine haemoglobinuria, are assuming smaller and smaller proportions with our increase of knowledge, and are probably non-existent when quinine is taken with perfect regularity.

5. "That there is at present but one preparation of quinine suitable for administration to young children—the tannate—and that it is best administered made into a sweetmeat with chocolate. (This latter suggestion unfortunately cannot be utilised under Indian climatic conditions.)"

The report itself is divided into two portions: Part I. dealing with epidemiology and Part II., which is much the larger, setting forth Celli's views on prophylaxis.

Some paragraphs in Part I. are devoted to the question of agriculture and malaria and Celli states that "more rational cultivation in marshy localities limits or reduces anophelic influence only in part, and though it proves beneficial—whether because of better nutrition arising from more abundant and varied products of the soil or because of the possession of house and clothing, with a less precarious mode of life—it fails notwithstanding by such indirect means to preserve man from malaria unless prompt and immediate medical, especially quinine prophylaxis, comes to the rescue."

It has been found that, whereas in Sardinia miners are more affected than agricultural labourers, the reverse is true in Sicily. This may be so because the lead poisoning to which the Sardinian miners are more or less subject predisposes them to malaria. Some of Celli's own conclusions terminating Part II. may be quoted. He says:—

(a.) "Since we have as yet no prompt and certain means of diagnosing latent infection, those parasitic forms which subserve the conservation of species in man and mosquito (manage to survive and) are still . . . absolutely resistant to quinine and every other remedy. No remedy therefore, quinine not excepted, is always effective . . . and so not even with the most accurate inter-epidemic and pre-epidemic treatment . . . can we believe it possible for malaria to be exterminated in an extensive locality already overrun by the disease.

(b.) "The *Consensus Omnium* to-day places quinine prophylaxis in the first rank amongst anti-malarial measures not alone for civilized countries but even for the colonies.

(c.) "In our more intensively cultivated levels, no less than in those under broad cultivation, the anti-anophelic campaign (except by the protection of the houses of a few of the better off against mosquitos) is not practically feasible. Mixed prophylaxis (anti-plasmodic and anti-anophelic) on the other hand, where . . . it can be simultaneously carried out by the administration of quinine and the provision of wire gauze netting to keep mosquitos at a distance from dwellings, conduces to reduce malaria to a minimum and to render it as mild as can possibly be wished."

The other conclusions chiefly concern Italy but there are many interesting facts in the report, which is well furnished with statistics and which points to the good results achieved by the quinine methods in Cochin China, Algiers, Greece (Anchiolos) and amongst the personnel of the German Navy.

A. B.

CELLI (A.). *Die Malariaabnahme in Italien.* [Decrease of Malaria in Italy.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. July 17. Vol. 75. No. 1. pp. 123-146.

Celli begins his lengthy review with a brief historical account of the Roman Campagna and the malign influence which malaria has had upon it and its inhabitants. He then describes what has been accomplished at Cervelletta, the estate in the Campagna where the great anti-malarial campaign began. Personal and general mechanical prophylaxis was found to be impossible in the case of the peasants while efforts directed against mosquitoes were only partially successful as, indeed, the author holds they must be throughout Italy. It was not till systematic quininisation was adopted that real progress was made. He goes on to speak of the malaria research stations throughout Italy and considers malaria in that country from an epidemiological standpoint. Latent infection carriers are found amongst both children and adults and, despite the most careful treatment, children relapse in the proportion of 38 per cent. and adults of 51 to 52 per cent. Childhood therefore is not specially predisposed to relapse.

For the general reader the section dealing with the research work by GAGLIO and his pupils as regards quinine will be found the most interesting and instructive part of the paper. Much that is stated is now well known but there are certain facts which merit attention. Thus it is noted that quinine hydrate or water-free quinine or the combinations which are insoluble in water, *e.g.*, the tannates, are absorbed just as well as the salts which are soluble in water. Again, although quinine is absorbed in the ordinary way, entering into a slight combination with hydrochloric acid in the stomach, a more important combination is with the bile acids and the carbon dioxide of the intestinal canal. So far as absorption goes it is immaterial whether the stomach is full or empty. Daily doses of quinine (medium — 30 to 40 centigrams) [roughly  $5\frac{1}{2}$  to 6 grains] can be tolerated longer and better than was at first believed. The ringing in the ears ceases after the first two or three days and it has been found that the drug, if properly given, is harmless and even aids nutrition.

Tolerance is readily established and quinine can be discontinued without any unpleasant results, while the fact of the organism being used to small or moderate doses in no way inhibits the action of curative doses, if these have to be given. It has been found that after the lapse of three days no quinine is to be detected in the urine, no matter what preparation is used or how it is given, and, as after a pause of more than three days in the administration, the disturbances which quinine causes again show themselves, it is better to give it daily or at least every second or third day. The salts which are insoluble in water are best tolerated because they are absorbed more slowly. The organotropic action of quinine, especially in sensitive individuals or those suffering from an idiosyncrasy or a tendency to black-water fever, is very restricted and ceases altogether if it is given in colloidal combination with tannic acid (so-called tannate), or with certain lipoids (lecithin, cholesterin). The tannate has been found better than the ethyl carbonate (euquinine) and di-quinine

carbonate (acistochnina) [? aristochinin], because these latter forms are always bitter and it is not easy to prepare them in a pleasant form, such as chocolate pastilles and the like. The tannate is also the least toxic of all the quinine salts and is especially useful in the case of children and in persons suffering from gastric and intestinal disturbances. The muriate and the bi-muriate are the best salts for general use.

The prophylactic method, employed first at Cervelletta, was to give throughout the summer and autumn two tablets (40 cgm.) daily to adults, one tablet to older children and a chocolate tablet with an appropriate dose to the smaller children. A great change resulted which was wholly attributable, according to Celli, to the quininisation. He passes on to deal with the special legislation now in force regarding malaria and the sale of quinine, and furnishes numerous statistical tables to prove the value of the quinine method. The article concludes with an account of the laws for improving hydraulic works (drainage of swamps, proper irrigation, clearing of canals, &c.) and of the statutes governing land improvement and settlement.

A. B.

**BENTLEY (C. A.). Quinine Propaganda.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 99-104. (1913. Simla: Govt. Central Branch Press.)

Although India consumes at present about one-sixth of the world's supply (17,000,000 ounces annually) of quinine, the vast proportion of her malarial stricken population receives no benefit from the drug. Much that is consumed is wasted owing to professional and lay ignorance while the existing methods of distribution have proved, more or less, failures. A system similar to that employed in Italy should be adopted, the widely advertised quinine being sold at a small profit and the revenue derived from the sale devoted to grants of quinine to dispensaries, &c.

The author believes the simplest curative method to be the administration of 20 grains a day for three weeks, in tablet form so long as the tablet is readily friable. Two sizes of bottles (the bottles being priced) should be adopted for general public use, one labelled "complete treatment" containing 126  $3\frac{1}{2}$  grain tablets, the other marked "relief treatment" holding 30 similar tablets. Full directions should be given as to dosage and the wrapper should contain a short history of quinine and an account of malaria. In conclusion the author urges an increase in cinchona plantations in India.

A. B.

**GRAHAM (J. D.). School Quininisation Experiments in the United Provinces.**—*Ibid.* pp. 105-111.

A record of the benefits which have resulted from a systematic quininisation both of teachers and scholars in schools throughout the United Provinces. Details as regards dosage and cost are given. In the case of children the author found no difficulty in administering uncoated tablets of quinine of from one-half to 5 grains. There was no compulsory administration.

A. B.

TURNER (J. A.). **Malarial Operations in the City of Bombay.**—*Ibid.* pp. 151-176.

Bombay, a city of 979,445 inhabitants and with, in addition, a large floating population, a total area of 22.53 square miles and an average annual rainfall of 72 inches was visited by a severe epidemic of malaria in 1908. As a result of a report on this outbreak by BENTLEY a special malaria officer was appointed and now the Medical Officer of Health reports on the methods which have been adopted to combat the disease and the difficulties which have been encountered in doing so.

The former have been very thorough. The Anophelines of Bombay are *A. rossii*, *barbirostris*, *listoni*, *stephensi*, *jamesii*, *culicifacies* and *fuliginosus*. With the exception of *A. rossii* all convey malaria. Attention however has not been confined to the mosquito aspect of the question as the following list of measures taken will demonstrate:—

1. Closing of wells and cisterns breeding larvae.
2. Treatment of all temporary pools and puddles with pesterine.
3. Giving a trial to fish before taking legal steps.
4. Distribution of quinine.
5. Filling in all hollows and low grounds with a view to remove potential sources of mosquito-breeding and to improve sanitation generally.
6. Improving of drains and gullies and extension of drainage schemes.
7. Improving of the general conservancy of the city.
8. Teaching the public by handbills and lectures with magic lantern slides.
9. Taking action in Court where parties defy the Municipality.
10. The spleen census of children and the examinations of blood of patients attending dispensaries.
11. A weekly return of cases of fever from all private and public dispensaries.
12. The examination of all species of mosquitoes found at different times of the year.
13. Proposed scheme for filling in a portion of foreshore and reclaiming another portion.

The difficulties in such a campaign are naturally very great. Details are given regarding the methods employed. With reference to well covers the author states that two kinds are allowed. One type is made of wire gauze, 20 meshes to a running inch. It is covered by expanded metal in order to protect the gauze underneath. With this sort of cover a trap door under lock and key or a hand pump is allowed if the water is required for religious observances, washing and similar purposes. These covers cannot last long on wells in open compounds as the force of the monsoon rains is apt to break the gauze which is also liable to rust. Hence there is a second type where the well is covered in with brick and cement except for a small opening of from 1½ to 2 square feet guarded by a trap door either of wire gauze or wood. These trap doors have to be kept under lock and key and opened only when water is actually being drawn. A leaflet giving hints for the prevention of malarial fevers is reproduced.

[The hints are excellent but the statement under No. 5 to the effect that "the well should be protected by a close fitting cover,"

would be improved by the addition of the words "and such covers must be frequently inspected and kept in good repair to the satisfaction of the sanitary inspector."]

A. B.

GRAHAM (J. D.). **Progress of present Anti-Malarial Schemes in the United Provinces.**—*Ibid.* pp. 177-182. With 3 maps.

The towns of Nagina, Saharanpur and Kosi in the United Provinces having been malarially surveyed and reported upon were inspected by special committees in May 1912. A report and estimate were furnished and then the Irrigation Department prepared detailed estimates of their projects. This was necessary as the Nagina project deals chiefly with tanks, water-logging and wet cultivation, the Saharanpur project with defects due to over-irrigation by canals, water-logging and wet cultivation and the Kosi project with irrigation, deficient drainage and water-logging.

Briefly the measures employed consisted in the abolition of rice cultivation within half a mile of the site dealt with, the limiting of canal irrigation, the flushing of canals, the altering of canal levels, the water-proofing of canal beds, the filling in of tanks and the regulation of borrow-pits.

[The report must be consulted for details, but it is a useful one for in the past it has not been easy to obtain the kind of particulars which it presents and in many countries irrigation works and anti-malarial campaigns are closely associated].

A. B.

BALFOUR (Andrew). **A Year's Anti-Malarial Work at Khartoum.**—*Jl. Trop. Med. & Hyg.* 1913. Aug. 1. Vol. 16. No. 15. pp. 225-232. With 5 figs. and 1 map.

Few papers dealing with the epidemiological aspects of malaria in Africa have appeared recently, so the author believes it may be of interest to give an account of the anti-malarial work in Khartoum for the year 1912. This year was an especially interesting one because the conditions favoured a prevalence of malaria, and Khartoum, which has enjoyed a very considerable immunity in past years from the disease, did not altogether escape, having been visited by a small epidemic following the occurrence of a short but heavy rainfall. This may have been due to the fact that very little attention has been paid, until recently, to the irrigated areas along the Nile to the North and far outside the municipal boundaries. These areas having gone from bad to worse began to constitute themselves a nuisance as nurseries for anophelines.

The local conditions of Khartoum, as regards malaria and mosquitoes, have already been considered fully by the author and they are therefore not specially dealt with again here. As regards the population a census was taken in the course of the year and the following are the official figures for the civil popula-

tion of Khartoum and Khartoum North, over which the Sanitary Service exercises control:—

**Khartoum City and Adjoining Villages.**

Europeans	...	...	...	...	1,114
Egyptians and other non-Europeans..					8,645
Natives	...	...	...	...	15,963
Total					25,722

**Khartoum North.**

Europeans	...	...	...	...	482
Egyptians and other non-Europeans..					1,467
Natives	...	...	...	...	15,176
Total					17,125

Central Prison (average)	...	...	...	...	363
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Grand total for civil population	...	...	...	...	43,210
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In addition the British and Egyptian Army garrisons amount to 5,507 men, 780 of these being British troops.

A series of tables give meteorological statistics for 1912, details of rainfall at Khartoum for August 1912. mosquito statistics for 1912 in Khartoum and Khartoum North, and anopheline infestations where pupae or pupal shells were found in the same year. A very excellent map gives a plan of the town with the areas where malaria has broken out and their relationship to mosquitoes, while photographs illustrative of anopheline breeding pools and mosquito traps are added. 87 cases of locally acquired primary malaria were notified throughout the year; a table shows the monthly distribution of these cases; 62 occurred in Khartoum and 25 in Khartoum North. There were no deaths amongst those attacked, the only fatal case recorded having acquired his infection outside Khartoum. Possibly some cases occurred amongst the native population but were never returned. A series of cases in December was rather interesting. These occurred amongst British troops and a most careful search, with the free use of traps, failed to reveal any anopheline infection of the barracks. Further, the anopheline infested area at Buri had been put out of bounds, while there was nothing to show that infection had been derived when the men were in camp on the main Nile. The most possible explanation was that they were recrudescences of slight or masked cases, relapses taking place as the cold weather set in.

It is pointed out that there is nothing to prevent another season of heavy rain and hence that it is very essential that levelling up and drainage operations should be taken seriously in hand. Some of these present difficulties from the engineering standpoint and there is the usual scarcity of funds. As regards the use of larvicides petroleum has been found to have its limitations, for in pools exposed to high winds it gets blown aside leaving an

untreated water surface on which mosquitoes readily lay their eggs. Sanitas-okol answers admirably and recently izo-izal has been tried with a fair promise of success. Creosote does not seem to have any special virtue to commend it. Experiments have also been carried on with traps. Figures are given showing the type of these used in houses; they are based on the lines of those advocated for sandflies and consist of wooden boxes lined by a dark material or painted black inside. A hinged door closes the trap below and in this there is a hole, which can be closed by a metal slide, and through which fumigation can be conducted. These traps have so far proved useful and Balfour thinks would be certainly valuable, given any great prevalence of anophelines.

Stress is specially laid upon the danger of irrigated areas and on the part they play as attraction centres. When the Gezira to the south of Khartoum is irrigated on a large scale the Sudan Government will be face to face with a very serious problem, but the author believes that with due care and the wise expenditure of the necessary funds, together with the stringent enforcement of existing rules and regulations the problem can be solved.

The lesson which has been learnt in Khartoum is that in anti-malarial warfare the motto must be defiance not defence. It is necessary to go out to the attack, not to wait to be attacked.

[This is a very interesting and instructive paper and should be carefully studied by all those who are engaged in anti-malarial work both in damp and dry climates throughout the world.]

G. C. Low.

WHITE (J. H.). **Malaria in Louisiana.**—*New Orleans Med. & Surg. Jl.* 1913. Aug. Vol. 66. No. 2. pp. 106-107.

The author proposes a scheme for concerted action between practitioners and the State Health Officer for the purpose of detecting and eventually wiping out malaria in Louisiana. He believes that many diseases, such as typhoid fever, &c., are at present erroneously classed as malaria owing to ignorance or slovenly diagnosis. He believes that if the measures he proposes are adopted complete success in the anti-malarial campaign will be obtained within a few years.

A. B.

ENGELAND (O.). **Meine Erfahrungen bezüglich der Malariaprophylaxe an Bord eines Kriegsschiffes.** [My Experiences with reference to Malaria Prophylaxis on Board a War-ship.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Aug. Vol. 17. No. 15. pp. 523-531.

This is an interesting account of a very thorough malaria prophylaxis carried out under the author's supervision on board a survey ship of the German Navy. The "Möwe" was for a period of 3½ months off the coast of South West Africa and called at many malaria-ridden ports. Engeland, as medical officer in charge of the ship, seized the opportunity of testing fully the



value both of mechanical protection and of quinine prophylaxis. He points out that what may be termed "personal" mechanical protection, *i.e.*, protection of the exposed parts of the body by veils, gloves, &c., is quite impracticable on board ship as it causes discomfort and interferes with work. It is impossible completely to protect a ship by mechanical means but these should supplement quinine prophylaxis. Before the latter came into general use the sickness and mortality returns from vessels of the German Navy employed on foreign service were very high. Latterly there has been a great change for the better, not only in the malaria rate but in the general sickness returns. Cases of black-water fever and fatal cases of malaria are now very rare. There is still, however, evidence that quinine prophylaxis is not properly carried out. The author explains his procedure, which gave most gratifying results. He believes in employing both prophylactic measures. He began the systematic quinine prophylaxis seven days before reaching the first port where malaria was known to be prevalent and based his method of administration on GRIEMSA's statement that after 24 hours most, and after 72 hours all the quinine which has been taken is excreted. His object therefore was so to arrange matters that the men under his charge should always have quinine present in their circulating blood.

He employed the hydrochloride and in bad localities gave it every third day. When the ship was at sea or in harbours where malaria was not so rife the quinine was given every fourth day, the dose being one gramme. In his presence and under the direction of the officers of the watch nearly every man of the ship's crew took a tablet containing 0.5 grammes every second day at bedtime. The dose was washed down with acid solution to ensure its being absorbed. This was carried out from the middle of June to the middle of November, 1911. Men of the middle watch who were on duty between 12 p.m. and 4 a.m. took 0.5 grammes at night and the same dose next morning. There were a few exceptions, *i.e.*, in the case of stokers, &c., who had specially trying work.

Officers were treated by NOCHT's fractional method [see paper by JUSTI in this number], taking the quinine at three or four days interval like the men. The prophylaxis was steadily continued after leaving the last harbour known to be malarious but the intervals were lengthened somewhat. The treatment terminated at the end of the eighth week. A certain number of cases proved unsuitable for the administration of quinine. These were men with poor digestions, bad teeth, neurasthenics and those who showed signs of quinine intolerance. Men coming under the first three headings were, as far as possible, prevented from going on shore expeditions, while intolerance was tested for by an initial dose of one gramme.

Sometimes, from quite unknown causes, quinine prophylaxis fails to avert malaria. Hence the necessity for mechanical protection which in any case is a useful adjunct. It was found that mosquitoes were frequently brought to the ship in water-supply boats, native crafts conveying vegetables, bum-boats, &c.

The following methods were carried into effect.

1. Whenever possible the ship anchored  $1\frac{1}{2}$  to 2 sea-miles from the land.
2. Leave of absence on shore was only granted during the day. Men were forbidden to enter native huts. Any members of the crew who had been ashore at night were each given one gramme of quinine on their return to the ship.
3. Fresh vegetables coming on board were immersed in water and the mosquitoes which they frequently harboured were thereby killed.
4. Covered native boats were not permitted alongside.
5. On the side where water and provision boats lay, all doors and windows were closed. After the departure of the boats the side was thoroughly washed down.
6. An energetic destruction of any mosquitoes which found access to the ship was carried out.
7. If the vessel had to be nearer than a sea-mile from land mechanical protection of port-holes &c. by means of gauze netting was employed. Wire gauze is not recommended. In the humid atmosphere of the West Coast it rusts rapidly, it is not easily repaired and it is much dearer than the ordinary netting. The latter should be fixed to frames of iron or wood and these should be covered by cloth to which the netting can easily be stitched. As SCHILLING states the mesh should not exceed 2 m.m. The netting should be in position by 6 p.m.
8. Only such lights as were absolutely necessary were permitted when the ship was near land.
9. The watch on deck wore mosquito boots and were allowed to smoke.
10. Survey parties on shore were provided with mosquito nets.

As a result of this careful system only one man of the ship's company (number, unfortunately not stated) contracted malaria. He was a cook and specially exposed to infection owing to the nature of his work, as the author explains. The prophylactic quinine failed in his case but the paper certainly shows what can be done by care and thoroughness.

A. B.

GIEMSA (G.). *Das Mückensprayverfahren im Dienste der Bekämpfung der Malaria und anderer durch Stechmücken übertragbarer Krankheiten.* [The Mosquito-spray Method in the Campaign against Malaria and other Mosquito-borne Diseases.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 13. p. 456.

A note emphasizing the fact that the satisfactory experiments carried out at the Hamburg Institute as recorded by Giemsa in his previous paper [see this *Bulletin*, Vol. 2, p. 154] were obtained by the use of the pyrethrum preparation furnished by the Berlin firm of RIEDEL.\* It is necessary to draw attention to this fact as adulterated preparations are inert. Mention is also made of a reduction in the price of the fluid, *i.e.*, from 2.65 to 2.38 marks per kilogram. A little further notice may be accorded Giemsa's former paper in which he describes the types of apparatus on the market.

These are made by the firm of Leonhard Schmidt, Grosser Burstah 46, Hamburg and there are three models, the first costing nine marks, the second and third 60 and 150 marks respectively. Model 1 is a hand-spray like a large garden syringe

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\* Address—J. D. RIEDEL A.-G., Berlin-Britz, Riedelstrasse.

and has interchangeable nozzles of varying form which can be fitted to the mouth piece so that different types of spray can be produced. Model 2 is in the form of a cylinder which can be carried on the back of the operator and holds ten litres of the special fluid. It works automatically under a pressure of five atmospheres. Model 3 is of a similar type but is larger, the container holding 30 litres and having a wheel and handles attached to it so that it can readily be moved about from place to place.

Giemsa points out that the efficacy of the method was shown by experiments on hibernating mosquitoes in cellars, stables, &c., in Hamburg and he recommends its use on a large scale for native huts which are so commonly infested by infected anophelines. Gangs of men armed with these sprays should systematically spray all native dwellings in malarial regions once every seven days, as it takes from eight to nine days for the malarial parasite to complete its cycle in the mosquito. It is important that the operators should work down-wind so that any mosquitoes which may have escaped destruction will not be blown back into huts already cleared but will more probably fly into those yet untreated and there meet their fate in due course. The method has an advantage over that of Ross in that, by its use, definitely infected imagines are frequently killed whereas anti-larval measures are directed against non-infected individuals and hence act in a more indirect manner. Its value in other mosquito-borne diseases (yellow fever, filariasis, &c.) is mentioned and it is stated to be beneficial in freeing plants from vermin as the fluid does not damage vegetation; the spray may perhaps find a use in destroying mosquitoes sheltering in plants and bushes in the neighbourhood of European dwellings.

[Giemsa's method is now employed on board Nile steamers at the quarantine station, Khartoum.]

A. B.

#### EXPERIMENTAL.

da ROCHA-LIMA (H.) & WERNER (H.). Ueber die Züchtung von Malaria-Parasiten nach der Methode von Bass. [Cultivation of Malaria Parasites by the Method of Bass.]—*Arch. f. Schiff-u. Trop.-Hyg.* 1913. Aug. Vol. 17. No. 16. pp. 541-551.

After mentioning the work of NOCHT who, *in vitro*, was able to trace a partial development of malarial schizonts, i.e., up to, but not beyond, the stage of sporulation, the authors record their own attempts and observations in 11 cases of tropical malaria and 6 of benign tertian fever. Like Bass they found the parasite of the former more easy to cultivate than the latter and they also agree that the best results are obtained when dextrose is added to the medium and the tubes are kept at room-temperature. They were, however, unable to substantiate *in toto* the results of Bass [this *Bulletin*, Vol. 1, pp. 22-24] or of those who support him, namely ZIEHMANN and THOMSON [this *Bulletin*, Vol. 1, p. 490 &

p. 648, and Vol. 2, p. 9 and pp. 224, 225]. This is evident from the following summary which gives the gist of their findings:—

1. They are unable to confirm Bass's complete work but can, like others, assert that his technique is useful for favouring a further development of a generation of malarial schizonts.

2. In the majority of 15 cases investigated they were able by Bass's method to trace this development up to the stage of division.

3. They failed to find evidence *in vitro* of any fresh commencing spore-forming cycles after the first division. They certainly saw in many cases, as long as 48 hours after the cultures were made, apparently vigorous, living, more developed forms of tertian and tropical [sub-tertian] schizonts, but these were traceable to a retardation of schizogony in the culture and did not proceed to a new second or third sporulation. They believe that Bass and the other observers mentioned drew faulty conclusions from what they saw and think that their own explanation is much more probably the correct one.

4. They could not observe a definite increase in the number of parasites. An increase is, however, easily simulated owing to the agglutination of infected red cells in the blood culture.

5. As noteworthy morphological peculiarities of the culture forms in tropical malaria they mention the fading of the erythrocytes which harbour the schizonts, a point already observed by THOMSON, and the premature clumping of pigment in the schizonts which is usually complete before the division has commenced.

A. B.

BROWN (Wade H.). Malarial Pigment (Hematin) as an Active Factor in the Production of the Blood Picture of Malaria.—*Jl. Experimental Med.* 1913. July 1. Vol. 18. No. 1. pp. 96-106.

The author refers to his previous experimental work\* on hematin intoxication in the rabbit, which he showed to be characterised by the production of a paroxysm analogous to that of malaria. In this paper he considers the effect of the pigment not only on the formed elements of the blood but also upon the coagulation time and the bleeding time. Rabbits were again used and the solvent for the hematin consisted of a solution of 1 or 2 per cent. bicarbonate of soda in 0.85 per cent. sodium chloride solution. Control experiments were conducted to exclude any fallacy resulting from the action of the solvent itself. It was found that though the red blood corpuscles are less subject to the toxin action of hematin than either the leucocytes or the platelets they may yet be markedly reduced in numbers by its intravenous injection. The mechanism of destruction is not clear. *In vitro* haemolysis does not follow the addition of the alkaline hematin solution. This was to be expected in view of the hypertonicity of the solution. It frequently does occur in the animal body but does not depend entirely on the amount of hematin injected. The most marked effect is on the platelets, which are rapidly destroyed, and associated with this destruction there is often a tendency to persistent and even profuse haemorrhage. The other effects induced are sufficiently indicated by the author's summary from which it will be seen that he regards hematin as an active factor in the production of many, if not all, the important changes in the blood that characterise

\* *Journal of Experimental Medicine*, 1912. Vol. 15. p. 579.

the various forms of malaria. In his opinion it is probably the hitherto unknown circulating toxin to which malarial anaemia has been attributed and he points out that the haemoglobin, converted to hematin by the malarial parasite, is not readily available for the regeneration of red cells and the blood is thus rapidly depleted of haemoglobin and iron, a condition tending to perpetuate the anaemic state. His summary is as follows:—

1. "Intravenous injections of alkaline hematin in the rabbit produce an anemia the severity of which is proportional to the amount of hematin injected and the susceptibility of the animal.

2. "Hemoglobinemia is an occasional consequence of hematin poisoning.

3. "The leucocytes in hematin intoxication are usually increased in number and are always characterized by a high percentage of large mononuclear cells and by pigmented phagocytes

4. "The platelets are markedly reduced by alkaline hematin and ultimately a prolongation of the coagulation time of the blood and of the bleeding time results.

5. "The anemia, the hemoglobinemia, the high percentage of large mononuclear leucocytes, the destruction of platelets and the tendency to haemorrhage in malaria are all influenced by the malarial pigment hematin."

A. B.

#### MOSQUITOES.

STRICKLAND (C.). **Short Key to the Identification of the Anopheline Mosquitoes of Malaya for the Use of Medical Officers and others.**—15 pp. 1913. Kuala Lumpur, F.M.S. Government Printing Offices.

According to the author there are for all *practical* purposes 15 species of Malayan anophelines. Hitherto in the case of these, as of other mosquitoes, it has only been possible to determine the species by the use of elaborate synoptic tables. Strickland, however, has arranged a simple and ingenious key whereby the Malayan species may readily be determined by medical officers, sanitary board officers and planters even when an ordinary magnifying glass is alone available. It can only fail if a new species be discovered but he thinks its advantages outweigh this objection. In a few words the *exclusive* descriptions of each species are stated and rough drawings help the reader. Thus in one species attention is directed to the tufted abdomen, in several others certain characteristics of the hind-legs are alone sufficient for determination, in yet others the hind-legs and the proboscis are taken together. A group of four, including three *Mysorhynchus* and a *Neostethopheles* can be distinguished from a study of their wings. The female palps form a guide to some, while, in others, the male palps plus the hind-legs furnish the clue. A note on the method of examination and a descriptive diagram of an anopheles, giving the male and female characteristics, serve as an introduction.

[While the key will doubtless fill a felt want, very few tropical sanitarians will agree with the opening paragraph of the author's preface which runs as follows:—"One can certainly eradicate malaria by wiping out all mosquitoes, for instance by a system of underground drainage a careful screening or destruction of all water holders, but why do this, when if one destroys these species

which cause malaria one gets the same result without having paid as well for the destruction of those which do no harm." It would have been better also if the illustrations of hind-legs on Plate VI had been drawn to a larger scale.]

A. B.

**Mosquito Device.**—*Canal Record.* 1913. June 11. Vol. 6. No. 42. p. 353.

An account of an ingenious contrivance for detecting the direction of the flight of mosquitoes. It is the invention of Mr. E. F. QUIMBY, an inspector in the Department of Sanitation, Canal Zone, Panama, and consists of a metal frame holding four plates of glass, each 12" x 12" in size, placed upon a tripod. These plates are held stationary at right angles to each other so that they are directed to the four points of the compass. To catch the mosquitoes they are smeared with a coat of tanglefoot made by adding half a pound of resin to a litre of castor oil. When smeared on the glass this coat is practically transparent. An all-night experiment with the apparatus showed "that the adult culex and anopheles flew on the quarter of a 17 to 19-mile wind; that the evening flight occurred between 6.10 and 7.10; that there was a complete lull in the flight after 7.10 o'clock; that there was a return flight between 5.50 and 6.40 in the morning, also on the wind's quarter; that the mosquitoes fly much higher and swifter in the morning than they do at night; and that, with possibly few exceptions, only adult females are in flight."

During another all-night test a single male anopheles was caught on the opposite side of the plates from the direction of the general flight. It is believed that it may have been flying about aimlessly when caught. "The instrument has proved to be useful in locating breeding places by eliminating the area facing the plates that do not show a catch. It also identifies the species of mosquitoes in the dark, as well as in the light, thereby determining the direction of flight, if any, during the night. Experiments show that there is no direct flight during the night, after a certain period. Further, this is the first time that a return flight has been recorded on the Isthmus; and it was shown to take place within a period."

An experiment tended to show that anopheles may fly low, slowly and in a zigzag flight as if seeking lulls in the wind.

A. B.

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## SLEEPING SICKNESS.

MACFIE (J. W. Scott). On the Morphology of the Trypanosome (*T. nigeriense*, n. sp.) from a Case of Sleeping Sickness from Eket, Southern Nigeria.—*Ann. Trop. Med. & Parasit.* 1913. Aug. 11. Vol. 7. No. 3 A. pp. 339-356.

Sleeping Sickness was first discovered in the Eket district of Southern Nigeria early in 1912; by the end of April, 1913 a total of 167 cases had been identified. The disease does not seem to be of a virulent type in Nigeria and does not occur in epidemic form. To account for this fact it has been supposed that the disease must have existed for a great number of years and that the natives must have acquired a relative immunity.

Two phases of the disease are recognised: an early phase characterised by enlargement of the glands of the neck, and a later stage in which lethargy appears. Cases in the former stage are common, but those that have advanced to the latter stage are comparatively rare. The disease appears to be mild, although a number of deaths are reported to occur annually. The duration is a matter of years. The great majority of the patients are from 6-18 years of age; the sexes are about equally affected. As a rule the general health is good, there is no marked anaemia or wasting. Irregular fever occurs and is accompanied by the general symptoms of febrile disturbances. Skin rashes—urticaria and erythema—are present in many cases; but they are also common in uninfected natives. Impotence and amenorrhoea are said to accompany the disease. Enlargement of the cervical and axillary glands is a constant symptom. The natives believe that the disease is curable by excision of the glands, an operation which their 'doctors' readily perform. Numerous healthy individuals bearing scars on their necks are to be met with who have, it is affirmed, been cured in this manner. According to the natives the glandular phase of the disease lasts for about four years before lethargic symptoms develop. From the fact that somnolence is not a common symptom, most of the patients appearing to be in the early stages, it is possible that many of them recover before this stage is reached. Trypanosomes have been found in the gland juice of practically every case, but up to the present they have not been detected in the peripheral blood.

A brief description of the Eket district is given. The towns are generally some distance from the water. The children and young adults are the water carriers, this possibly accounting for the fact that they form the majority of the cases of sleeping sickness. Tsetse flies are prevalent all over the district; *G. palpalis*, *G. caliginea* and *G. tachinoides* have been found. Dr. FORAN has observed that the tsetse flies 'appear to follow pigs about more than any other animal, and it is generally easier to catch the flies where these animals are than at the water.'

*The strain of trypanosome.*—A long and detailed account of the morphology of the trypanosome, as it appears in a guinea-pig, is given. There do not appear to be any features by which it can be distinguished from *T. gambiense* on microscopic examination. It is a polymorphic parasite, the individuals of which vary in

length from 8.34 microns; the average length is 21.5 microns. Posterior nuclear varieties have not been encountered. A biometric investigation of the trypanosome was undertaken, and 100 individuals of the infection in the guinea pig were measured on each of 10 consecutive days. In a chart the curve of the trypanosome is compared with that given by STEPHENS and FANTHAM for *T. gambiense*, and in a table the average, maximum, and minimum lengths and the percentage of short, intermediate, and long forms of the two parasites are compared. The author remarks that it will be observed that the trypanosome from Southern Nigeria is somewhat shorter than *T. gambiense*; the average length of 1,000 individuals being 21.5 microns as compared with 24.9.\* The maximum and minimum lengths of this parasite are 34 and 8 microns as compared with 36 and 16 in the case of *T. gambiense*. The curve of this parasite is lower and more extended, and reaches its main peak at 21 instead of at 26 microns. The occurrence of extremely small individuals is characteristic of this strain.

*Pathogenicity of the trypanosome.*—Dr. FORAN at Eket inoculated a number of rats with the blood and gland juice of cases of sleeping sickness, but none of them became infected. Of six guinea-pigs only one had shown parasites in the peripheral blood to the time of writing; the infected animal was alive and well 124 days after inoculation. Of seven mice three showed trypanosomes in the peripheral blood after incubation periods of 4, 6 and 8 days, respectively; parasites had not been seen in the others up to the time of writing. Two goats were negative up to the 24th day, as were also two dogs. A monkey became infected on the 10th day and was alive and well on the 22nd day after inoculation.

In conclusion the author considers that this trypanosome differs in several respects from *T. gambiense*. In man it produces a form of sleeping sickness that is relatively mild, and occurs most commonly in young people. Trypanosomes are apparently either absent from the peripheral blood altogether, or present in such small numbers that hitherto they have not been detected. To the smaller laboratory animals the strain is but slightly pathogenic. The trypanosome is smaller than *T. gambiense* and there appear constantly in the blood films a few very minute parasites measuring as little as 8 microns in length. It is stated that the occurrence of a few peculiar trypanosomes which appear to have a flagellum free in their whole length is remarkable.

The author is convinced that this trypanosome from Nigeria cannot be regarded as belonging to the same species as *T. gambiense* and proposes for it the name *T. nigeriense*.

[The most important part of this paper is that dealing with the recognition of a chronic form of trypanosomiasis in children and young adults. The author has come to the conclusion that the chronicity is to be explained on the ground that the pathogenic agent is a new human trypanosome. The evidence he advances to support this view is, however, inconclusive. The parasite was

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\* STEPHENS and FANTHAM's measurements were from rats.



examined in one guinea-pig only and the points of differentiation mentioned by the author would hardly justify a description of a new human trypanosome. The anterior displacement of the nucleus is seen in other trypanosomes and the flagellum 'free in its whole length' is also frequently to be observed in many species of trypanosomes and is probably an artefact. The chronicity of the disease might equally well be explained by the assumption of a special tolerance on the part of the host.]

W. Yorke.

HEARSEY (H.). *Nyasaland Sleeping Sickness Diary*. 1913. April 30. Part 20. 11 pp. Zomba: Government Printers.

An account is given of 20 additional cases of human trypanosomiasis reported during the period January to April, 1913. Nineteen of these were found in the sleeping sickness area and one near Kotakota in the Marimba district. Fourteen of the 20 patients have died, as have also cases 105 and 106. To the time of writing 128 cases of sleeping sickness had been discovered in Nyasaland. Clearing of bush and scrub is being carried out around all villages in the sleeping sickness area. A re-investigation of the districts to the north and south of the Proclaimed area is shortly to be undertaken in order to ascertain whether these localities harbour cases of sleeping sickness.

W. Y.

ECKARD (B.). *Ueber Schlafkrankheit*. [On Sleeping Sickness.] —*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 14. pp. 494-497.

The author states that the view is generally held that the onset of sleeping sickness is so extremely insidious that a person may be infected for months or years without the cognizance of either himself or the doctor. He quotes MENSE's handbook on tropical diseases in support of this statement.

As the result of his personal experience in German East Africa the author is convinced that sleeping sickness commences acutely with the most violent symptoms (*stürmischsten Erscheinungen*) about 14 days after the infective bite. Various cases (5 native, 2 European and 1 Indian) in which the approximate date of the infective bite could be determined are quoted in support of this contention. High temperatures and rigors were prominent symptoms. The results of treatment of these eight cases are peculiar. Whilst in the natives the trypanosomes disappeared after the first injection of atoxyl and the patients after one or two atoxyl treatments remained cured, the infections in the Indian and the two Europeans proved from the commencement to be atoxyl fast. Whether the strain was more virulent in the latter or whether these are more susceptible than the natives the author is unable to state. It might be mentioned that in Rhodesia and in the southern district of German East Africa the human infections appear to be rather atoxyl fast. The author goes on to describe the course of the disease.

W. Y.

## UNGULATES AND SLEEPING SICKNESS.

KLEINE (F. K.) & ECKARD (B.). Ueber die Bedeutung der Haustiere und des Wildes für die Verbreitung der Schlafkrankheit. [On the Significance of Domestic Stock and Game for the Spread of Sleeping Sickness.]—*Zeitschr. f. Hygiene u. Infektionskr.* 1913. July 17. Vol. 75. No. 1. pp. 118-122.

These investigations are a continuation of the work of KLEINE and FISCHER (*Sleeping Sickness Bulletin*, Vol. 3, p. 402). Of 13 experiments, in which infective *G. palpalis* were fed on healthy goats and sheep, 9 were negative, whereas in control experiments in which monkeys were used all became infected. These experiments indicate the difference of susceptibility of monkeys and domestic stock. [These results should be compared with those of DUKE (see this *Bulletin*, Vol. 2, p. 240).] Goats and sheep would appear to have but little significance as a reservoir of the infection. *G. palpalis* could not be infected on three of seven animals in which *T. gambiense* was found in the blood. The infectivity diminished with the length of time which had elapsed from the first appearance of trypanosomes in the blood.

The authors had the opportunity of examining two of these goats, 73 and 89, again. They were infected in September, 1910 and in March, 1912 no parasites were found in the blood. Several blood injections into monkeys were negative. From April 9 to August 3, 1912, a large number (744) of laboratory bred *G. palpalis* were fed on Goat 73 and subsequently on healthy goats. All the flies were examined, but no trypanosomes were found. The goats were hence cured. Goat 73 was reinoculated from a sleeping sickness monkey, but did not become infected; after a second reinoculation a single trypanosome was once found in its blood. Goat 89 became ill after the first reinoculation and again after the fourth, but subsequently recovered. Resistance and immunity against *T. gambiense* were further demonstrated. A number of goats, sheep and cattle were repeatedly inoculated with virulent blood. Only 10 of 21 animals became ill after the first injection, 5 after the second, 4 after the third, 1 after the fourth and 1 remained healthy. Parasites were only rarely found in the blood of infected goats and in small numbers. Often the incubation was very long—in one sheep over 46 days. The experimental animals always remained in good condition and exhibited no signs of disease.

The authors examined 113 mammals from sleeping sickness districts on Tanganyika; 15 cc. of blood from the jugular vein or heart were injected into healthy monkeys. Twenty-three bushbuck, one hippo, and four bushpig were found to be negative. Trypanosomes (*T. cazalhoui* or *T. nanum*) were found in the blood of two bushbuck. Five cattle, 55 goats and 25 sheep were examined from districts heavily infected with sleeping sickness and of these 1 ox, 1 goat and 1 sheep (i.e., 3.5 per cent. of the domestic stock or 2.5 per cent. of the total mammals examined) were found to be infected with *T. gambiense*. Parasites of the *cazalhoui* type were found in 3 oxen, 2 goats and 1 sheep.

As a result of these investigations the authors are of opinion that domestic animals play a much less important part as reservoirs of sleeping sickness than human beings. Although the authors did not find *T. gambiense* in game, yet DUKE has shown that natural infections do occur in antelopes. General measures are, however, unnecessary on account of the relatively small percentage infected, especially in those districts where the disease is mainly spread by *G. palpalis*. Whether and where the game should be destroyed must depend on local conditions. The chief significance of the game as parasite carriers lies, in the authors' opinion, in the fact discovered by BRUCE that in regions from which the human inhabitants have been completely removed on account of sleeping sickness the infection has not died out after several years.

With regard to *T. rhodesiense* the circumstances are not so clear, as this parasite is at present indistinguishable from the cattle trypanosome *T. brucei*. The view of KINGHORN and YORKE that in the Luangwa Valley of Northern Rhodesia 16 per cent. of the game is infected with *T. rhodesiense* is extremely improbable, as they failed to find the morphologically similar and widely spread *T. brucei* in such a definite *morsitans* country.

The authors have considered the question as to whether *T. brucei* may not exceptionally become virulent for men and whether the insusceptibility of adults may not be due to slight infection during childhood. They have so far found no support for this supposition; about 500 children were examined in *morsitans* districts without finding any case of trypanosomiasis. [The authors are evidently unaware of the opinions expressed by the Royal Society Commission to Nyasaland and others that *T. rhodesiense* is identical with *T. brucei* or *T. ugandae* (STEPHENS and BLACKLOCK).]

W. Y.

TAUTE (M.). Untersuchungen über die Bedeutung des Grosswildes und der Haustiere für die Verbreitung der Schlafkrankheit. [Investigations on the Significance of Big Game and Domestic Stock for the Spread of Sleeping Sickness.]—*Arbeit. a.d. Kais. Gesundheitsamt.* 1913. Aug. Vol. 45. No. 1. pp. 102-112. With 1 plate.

In view of the great divergence of opinion as to the practical significance of big game and domestic stock in the campaign against sleeping sickness these investigations were undertaken especially with a view to examining the work of KINGHORN and YORKE, who found that in the Luangwa Valley of Northern Rhodesia 16 per cent. of the game were infected with *T. rhodesiense*.

The experiments were carried out at Lubimbinu in Portuguese Nyasaland about 90 km. from Fort Johnston and about 600 m. above sea-level. Lubimbinu is itself fly-free, but is surrounded by a large wooded fly belt in which *Glossina morsitans* was present in enormous numbers and in the rainy season Tabanids were plentiful. The population is free from trypanosomiasis, but quite

the reverse is the case with domestic stock. Cattle and sheep are not found, goats are rare, and dogs are very seldom seen and rapidly die.

The experimental animals were brought from fly-free areas in fly-proof boxes and only used after a sufficient period of quarantine; monkeys and dogs were chiefly used.

*Rhodesiense-like trypanosome in big game.*—From the end of August until the end of December, 1912, 37 wild animals, including buffalo (1), eland (1), zebra (2), waterbuck (10), hartebeest (12), bushbuck (3), reedbuck (2), duiker (4), warthog (1) and bushpig (1), were examined in the above district. The results of direct examination of the blood and of the subinoculations into monkeys, dogs and goats are given in a table. Trypanosomes were found by direct examination in 19 instances. Of the 25 subinoculations made into monkeys and dogs 6 were positive and of the 3 made into goats 1 was positive. Of the 37 animals examined trypanosomes were found either by direct examination or by subinoculation or by both methods in 20. Trypanosomes similar morphologically, and as regards their pathogenicity, to *T. rhodesiense* were found in 6 of the subinoculated monkeys and dogs. The rest of the trypanosomes found in the game belonged to the groups *T. vivax* and *T. pecorum*.

*Rhodesiense-like trypanosomes in domestic stock.*—In 369 wild *Glossina morsitans* examined mammalian blood was found in 69 (18·7 per cent.) and developmental forms of mammalian trypanosomes in 27 (7·3 per cent.). In 10 experiments 1,069 wild *Glossina morsitans* were fed in batches on 8 dogs and 2 monkeys and 6 of the animals became infected with a polymorphic trypanosome exhibiting posterior nuclear forms in 4-6 days. Seven dogs and one goat were purchased in the villages situated in the fly belt and all were found to be infected with a polymorphic trypanosome exhibiting posterior nuclear forms. Two other goats were found to be infected with a trypanosome non-pathogenic for monkeys and dogs which was probably *T. vivax*.

The author gives a detailed description of the trypanosome found in the antelope and domestic animals. It is a polymorphic parasite exhibiting posterior nuclear forms and indistinguishable morphologically from *T. rhodesiense*. The maximum, minimum and average lengths of 2,000 individuals were 36, 13 and 21·8 microns, respectively. These figures are compared in a table with those obtained by BRUCE, STEPHENS and FANTHAM, and KINGHORN and YORKE for *T. rhodesiense* from men and with those obtained by KINGHORN and YORKE for the antelope strain of *T. rhodesiense*. The biometric curve obtained by the author is also compared with those of KINGHORN and YORKE, and BRUCE and his colleagues.

The pathogenicity of the trypanosome was examined and the average duration of the disease was in 36 dogs 17 days, in 12 monkeys 23 days, in 9 goats 16 days and in 5 rats 24 days. Three oxen were still alive on the 29th day, but all showed signs of disease. Infected dogs and goats showed interstitial keratitis and a characteristic oedema of the face.

*Experimental separation of the antelope and domestic stock trypanosome from true T. rhodesiense.*—The author remarks that if the view of KINGHORN and YORKE be correct that the trypanosome in question is really *T. rhodesiense*, then in this locality 16·2 per cent. of the game and an almost equal proportion of domestic stock were infected with it in spite of the fact that no case of human trypanosomiasis has been discovered in the district. Thus the game would here not only act as a reservoir, but also as a precursor of Sleeping Sickness. To prove that this view is erroneous the author performed the following experiment on himself. Ninety-three laboratory-bred *Glossina morsitans* were fed for 4 days on a monkey infected with the antelope strain and after starving for a day were fed on a series of healthy animals, as in the table.

TABLE III.

No. of flies.	Flies fed		Result.	Course of disease.	Remarks.
	From	On			
74	6th-12th day	Goat No. 16...	Goat remained well.		
61	18th-16th "	Goat No. 22...	" "		
52	17th 19th "	Goat No. 34...	" "		
45	20th-22nd "	Goat No. 43...	Goat developed trypanosomiasis.	Died on 20th day of infection	
89	23rd-25th "	Monkey No. 75	Monkey developed trypanosomiasis.	Died on 25th day of infection	
84	26th-27th "	Dog No. 51 and Dog No. 76.	Both dogs developed trypanosomiasis.	Dog No. 51 became blind on the 16th day and died on 19th day of infection. Dog 76 showed keratitis on 14th and died on 24th day of infection.	Well-marked posterior nuclear forms found in blood.
82	28th-32nd "	Man (TAUTE)	Man remained well.	—	
81	33rd-35th "	Monkey No. 72	Monkey developed trypanosomiasis.	Died on 14th day of infection.	
29	36th-40th "	Dog No. 71...	Dog developed trypanosomiasis.	Died on 8th day of infection.	Well marked posterior nuclear forms found in blood.
27	41st-44th "	Dog No. 33...	Dog developed trypanosomiasis.	Showed keratitis on 18th day and died on 21st day of infection.	

In a second and similar experiment 77 laboratory-bred flies were allowed to feed for 4 days on 5 animals infected with 5 different antelope strains; the subsequent procedure was similar to that described above. The author did not become infected.

In a further experiment 2 cc. of the blood from a naturally infected dog was injected into the author and also in various amounts (2 cc. or less) into a monkey, 3 dogs, a goat and 2 rats. The author remained healthy whilst the experimental animals all became infected and died.

These experiments show that a trypanosome, from wild game and domestic stock, indistinguishable from *T. rhodesiense* does

not infect man either when infected flies are allowed to feed upon him or by direct inoculation of the infective blood. Reference is made to the work of WENYON and BLACKLOCK who showed that posterior nuclear trypanosomes were to be found in *T. peccaudi* and *T. brucei*. The author considers that the game and domestic stock trypanosome is *T. brucei*. He refers to the somewhat similar experiment recorded by TODD (*Sleeping Sickness Bulletin*, Vol. 3, p. 174)\* in support of this opinion. In order to demonstrate that he was not infected the author inoculated 90 cc. of his blood in doses of 2-15 cc. into a monkey and 7 dogs; these did not become infected. The paper concludes with the remark that *T. brucei* cannot live in the human body.

The conclusions are:—

Trypanosomes in naturally infected game and domestic stock can only be regarded with certainty as the cause of Sleeping Sickness when they have been shown to be pathogenic for man.

The game and domestic animals do not at least play the part in the spread of sleeping sickness supposed by KINGHORN and YORK.

In judging of the infectivity of a region with Sleeping Sickness it is not sufficient merely to prove that the bite of the wild *Glossina* gives rise to an infection in experimental animals with an apparently identical trypanosome.

In the east Nyasaland area *T. brucei* can be distinguished from the pathogenic agent of Sleeping Sickness only by the fact that it is non-pathogenic for man: it rapidly dies in the human body.

[These experiments are obviously of great value, but it is doubtful if they establish the point which the author wishes to prove, namely, that the antelope trypanosome is different from the human parasite, *T. rhodesiense*. All the author has proved is that a healthy human individual has been able to resist infection. A possible interpretation of the facts is that at the present time the normal human being is able to offer considerable resistance to infection with *T. rhodesiense* and hence it is only occasionally that a case of infection occurs.]

W. Y.

#### TREATMENT.

KOLLE (W.), HARIOCH (O.), ROTHERMUNDI (M.), & SCHÜRMANN (W.). **Chemotherapeutische Experimentalstudien bei Trypanosomen Infektion.** [Chemotherapeutic Experimental Studies in Trypanosomiasis.]—*Zeitsch. f. Immunitätsforsch. u. experiment. Therapie*, 1913. Aug. 5. Vol. 19. No. 1. pp. 66-97.

Most of the work recorded in this paper has been published previously (see this *Bulletin*, Vol. 2, pp. 134 and 249). In these papers, which dealt with various preparations of antimony as therapeutic agents in trypanosomiasis, it was pointed out that "Trixidin" (antimony trioxide) was of remarkable value. It

\* This experiment, in which blood from a trypanosome infected cow was injected into a European and two natives without untoward effect, differed in that there is nothing to show what the species of trypanosome was. It might have belonged to the *ricar* or *pecorum* group.—A. G. B.

was practically non-poisonous in the doses used and of great therapeutic efficacy. In a table is shown the effects of large doses of trixidin on normal mice. It was found that 240 mgm. of the drug killed mice of 15 and 17 gm. on the 2nd and 3rd day, respectively, but a dose of 120 mgm. per 10 gm. of body weight was well borne by the animal and no symptoms of acute or chronic poisoning resulted. In another table is given the result of treating 12 mice, infected with nagana, with trixidin. In this series of experiments it is shown that 1 mgm. of the drug is sufficient to cure the mice and prevent relapses.

Experiments were also carried out to ascertain whether trixidin had any prophylactic action against infection with trypanosomes. The results of these experiments are shown in tables. The drug prevented infection in a great proportion of cases and in certain animals infection did not occur when they were inoculated with a virulent strain of nagana sixteen days after the drug had been administered.

The second portion of the paper deals with the cutaneous administration of insoluble antimony compounds in ointment form. The results of treating infected mice by systematic daily inunctions of 50 per cent. metallic antimony ointment, or with 40 per cent. dimethylphenylpyrazolonacethylantimony ointment are given in tables. By this method the authors succeeded in curing between 60 and 70 per cent. of mice infected with nagana, dourine, or *T. gambiense*.

In a postscript the authors write that they carried out experiments on animals infected with the spirochaete of African relapsing fever. These preparations of antimony whether administered intramuscularly or by inunction had no effect on this parasite. Similar negative results were obtained in the case of mice infected with *Schizotrypanum cruzi*.

In their conclusions the authors state:—

The toxicological actions of antimony compounds—soluble and insoluble—require further study, especially in regard to organotropy and chronic poisonous effects.

Antimony compounds are chemotherapeutically active only when they contain the antimony in the trivalent form. Antimony trioxide, "Trixidin," is of all hitherto known antimony preparations the most active in intramuscular injection. By one or two intramuscular injections of absolutely nonpoisonous doses of "Trixidin" 100 per cent. of mice infected with nagana can be permanently cured.

Metallic antimony also cures infected mice, but gives rise to chronic poisoning ending in death. Administered, however, in ointment form it is equally efficacious and is not poisonous.

The organic insoluble compound "Dimethylphenylpyrazolenantimony-chloride" is also, when administered in ointment form, a remedy which permanently cures without the slightest poisonous effects.

The formation of depots of insoluble, slowly absorbable compounds of antimony such as antimony trioxide acts prophylactically against infection with trypanosomes.

As a result of these experiments the treatment of chronic trypanosome infections by repeated administrations of insoluble, slowly absorbable, antimony preparations in combination with other remedies is recommended, in order to obtain through slow but continuous absorption a lasting action and permanent cure.

W. Y.

TANON (L.) & DUPONT (A.). *Traitement de la Trypanosomiasse humaine.*—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris.* 1913. May 15. 3 ser. Vol. 29. No. 16. pp. 975-988.

An account is here given of the therapeutic action of two new derivatives of arsenic—1116 (tétraoxydiphosphaminodiarsénobenzène) and 1151 (phényldisulfaminotétraoxydiaminodiarsénobenzène)—in trypanosome and spirochaete infections in experimental animals and man.

Experiments were carried out with rats, mice, guinea-pigs and *Macacus rhesus* infected with *T. gambiense*, and rats and mice infected with *Sp. duttoni*. The animals infected with *T. gambiense* were cured in two or three hours after a subcutaneous injection of 1116 or 1151. The drugs were given in the following doses:—mice .001 gm. per 10 gm. of body weight, rats .02 gm. per 100 gm. of body weight and monkeys .1 gm. per kilo of body weight. The injections provoked some slight local reaction which disappeared in 3 or 4 days. For subcutaneous administration the salt was dissolved in distilled water in the proportion of .05 gm. per 10 cc. In these doses the drugs caused the trypanosomes to disappear rapidly and no relapse was noted in any of the 500 animals used by the authors.

An account is given of the manner in which the parasites are destroyed. The phenomena were studied *in vivo* and *in vitro*: the disappearance of the trypanosomes is due to trypanolysis and not to phagocytosis. In the case of spirochaetes fragmentation precedes spirillolysis.

The value of the drugs was tested on sleeping sickness patients in Senegal. In all, 30 cases were treated and the authors are able to record 15 as definitely cured. Those probably cured are more numerous, but the lack of experimental animals has obliged the authors to delay publication regarding several of these. Only those cases were treated in which trypanosomes had been definitely found in the blood or glands. Four injections of 1151 or 1116 dissolved in 3 cc. of 12 or 15 per cent. sodium carbonate serum were given at intervals of 8 days; 25 per cent. carbonate serum was also employed, but such solutions caused vomiting. The drug was given intravenously, as subcutaneous or intramuscular injection caused considerable pain. Administration per os was found to be valueless. A month later the blood and gland juice were examined and if no trypanosomes were found 10 cc. of blood and 10 cc. of cerebro-spinal fluid were injected intraperitoneally into a monkey (*Cercopithecus ruber* or *patas*). If the monkey proved not to be infected at the end of 40 days the patient was considered to be cured. Details are given of six cases cured, the authors say, by means of 1116 or 1151. Treatment is easy and the drugs are well borne by the patients, nor do there appear to be any serious accidents following the injections. The vomiting and diarrhoea, sometimes noticed, may be attributed either to the drug itself or, as the authors are inclined to think, to the hyperalkalinity of the serum employed. These symptoms were more noticeable after administration of the 25 per cent. carbonate serum than after the 12 or 15 per cent.



These drugs were found to cure the disease in the 1st and 2nd stage, but not in the 3rd stage when the cerebro-spinal system is involved, as they penetrate but slowly into the cerebro-spinal fluid. Possibly injections of the diluted drug into the cerebro-spinal canal might prove successful, but experiments are necessary.

W. Y.

LAVERAN (A.) & ROUDSKY (D.). *Le Galyl dans les Trypanosomiasés*.—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 502-505.

The authors refer to the work of TANON and DUPONT (see above) which showed that two new arsenical compounds 1116, or galyl, and 1151, or ludyl, were of considerable therapeutic value in animals and man infected with *T. gambiense*. In the present paper the action of galyl was tested on animals infected with other pathogenic trypanosomes. Galyl is a yellow powder which is insoluble in water, but a solution is readily obtained if sodium carbonate is added to a fine suspension of the drug in distilled water. Solutions of the following strengths were injected intramuscularly—in mice and rats 1 in 200 and in guinea-pigs 1 in 100. There is only slight local reaction in the former animals, but in guinea-pigs the reaction is more marked, painful on pressure and lasts for several days. In mice of 20 gm. up to 1.5 mgm. can be given; in healthy mice 2 mgm. can frequently be given, but infected mice are more sensitive and 1.5 mgm. sometimes kills. This is probably due to the combined poisonous effects of the drug and toxins liberated from the disintegrated trypanosomes. In guinea-pigs 1 cgm. can be given for each 100 gm. of body weight. The drug was tested on animals infected with the following trypanosomes:—*T. brucei* (nagana ferox, EHRLICH), *T. evansi*, *T. rhodesiense*, *T. gambiense*, *T. soudanense*, *T. hippicum*, *T. dimorphon*, *T. congolense*, and *T. pecorum*. Galyl was found to be very active in infections of *T. brucei*, *T. evansi*, *T. rhodesiense*, *T. soudanense*, *T. gambiense* and *T. hippicum*. It was, however, quite inactive in the case of *T. dimorphon*, *T. congolense* and *T. pecorum*. Ludyl was equally inefficacious in the case of *T. dimorphon*.

The authors point out that the activity of a drug is often more marked on one species of trypanosome than on another, but it is exceptional to find such a clearly cut distinction between sensitive and refractory trypanosomes as occurs in the case of this drug. The fact that galyl is inactive for *T. dimorphon*, *T. congolense* and *T. pecorum* shows once more that these parasites are closely related.

W. Y.

RITZ (H.). *Chemotherapeutische Versuche mit "Trypasafrol."* (Zum Teil nach Versuchen von Frl. F. Leupold.) [Chemotherapeutic Experiments with Trypasafrol.]—*Berlin Klin. Wochenschr.* 1913. July 28. Vol. 50. No. 30. pp. 1387-1389.

The author refers to the work of BRIEGER and KRAUSE (see *Sleeping Sickness Bulletin* Vol. 4. p. 328) on treatment of experimental trypanosomiasis with a dye of the safranin series

called by them 'Trypasafrol.' The results of these workers appeared so satisfactory that Ritz decided to repeat the experiments. The technique employed was that of BRIEGER and KRAUSE. Infected animals were fed on food with which trypasafrol was mixed. In a further series of experiments, in order that the exact dose administered might be known, a solution of the dye was passed into the animals' stomach by means of a tube. Two strains of *T. brucei* were used, (1) MORGENROTH'S which is extremely sensitive to arsenic and exhibits a normal sensitiveness to dyes, (2) the Ferox strain of EHRLICH which is partially arsenic-fast.

The lethal dose of the dye was first ascertained. It was found that when the drug was administered by means of the stomach tube 0.5 gm. per kilo of body weight was fatal for guinea-pigs and 0.4 gm. was not always well borne. The lethal dose in mice was approximately 0.01 gm. per 20 gm. of body weight. About 0.06 gm. was found to be toxic when administered more gradually, mixed with the food.

The results of the experiments on mice, rats and guinea-pigs are given in four tables. Only a single mouse was cured. The author writes that trypasafrol does not cure mice, rats and guinea-pigs experimentally infected with the strains of *T. brucei* used by him. It appeared to exert a slight effect in mice, as the course of the disease was somewhat prolonged and one mouse out of 19 was cured. It was observed that 60-70 per cent. of trypanosomes in the treated animals lost their blepharoplast.

W. Y.

#### IMMUNISATION.

- (i.) RONDONI (Pietro) & GORETTI (Guido). **Studien über Schutzimpfung gegen experimentelle Naganainfektion.** [Studies on Vaccination against Experimental Nagana Infection.]—*Zeit. f. Immunitätsforsch. u. experim. Therapie*. 1. Teil. Orig. 1913. July 17. Vol. 18. No. 5. pp. 491-515.
- (ii.) **Ricerche Sperimentali sul Nagana.**—*Lo Sperimentale*. 1913. Aug. 16. Vol. 67. No. 4. pp. 427-453.

These papers are identical and give an account of attempts to produce active immunity in respect to infections by *T. brucei*. The authors express their conviction that in order to obtain successful immunity one should attempt to damage as little as possible the substance of the body of the trypanosome, to which the antigen property is bound. The trypanosomes must be killed or rendered innocuous in such a manner as to preserve the integrity of their receptors.

The authors adopted four procedures: treatment of the trypanosome suspension with (1) distilled water, (2) hypertonic solution of NaCl, (3) salts of quinine, and (4) salvarsan. White mice were used chiefly for the experiments. A virulent strain of nagana was employed and the trypanosome suspension obtained from rats by fractional centrifugation of their blood when heavily infected.

The administration of vaccines prepared by use of methods (1), (2) and (3) caused a definite prolongation of the incubation

period on subsequent (3-8 days later) injection of virulent trypanosomes and death occurred several days later than in the case of the controls. But by each method no absolute immunity was produced.

Vaccines prepared with salvarsan (Method 4) gave good results. The authors found that between the strengths of salvarsan which killed the trypanosomes outright, and that which merely weakens them without rendering them innocuous, there was an intermediate strength which although it did not cause their death, yet was sufficient to render them quite incapable of multiplying in the animal body. The experiments described show that there must be a definite quantitative relationship between the salvarsan and trypanosomes employed in order to obtain a successful vaccine. The salvarsan must be fresh and cannot be used after 10 or 15 days. Two large white rats were bled at the height of the infection, and the blood collected in citrate of sodium broth and centrifuged gently. The liquid containing the majority of the trypanosomes and a few red cells was mixed with a solution of salvarsan in such a manner as to make a dilution of salvarsan 1 : 40,000. The emulsion was kept half an hour at room temperature to permit the combination of the arsenical product with the trypanosomes. After this period the latter appear well preserved and mobile. It is advisable to choose such a dose of salvarsan as causes a slight slowing of movement, in order to be practically certain of the avirulence of the parasites and avoid accidental infection and loss of the animals. After strong centrifugation the sediment is taken up in a little salt solution. This constitutes the vaccine and is injected intraperitoneally into mice. If mice treated with this salvarsan-vaccine are later inoculated, many remain free from parasites (observation several weeks), others show parasites and die a long period after the controls. Forty to fifty per cent. of animals vaccinated a single time with this salvarsan-vaccine are completely refractory to subsequent inoculations with small doses of trypanosomes. A table containing the results of the experiment is given; 18 mice including 3 controls were used. The serum of rabbits treated with the vaccine possesses marked trypanocidal properties. The authors consider it desirable to extend these researches and apply them to other species of trypanosome, as such a method might give practical results of some importance.

W. Y.

SCHILLING (Claus) & RONDONI (Pietro). *Ueber Trypanosomen-Toxine und-Immunität*. [On Trypanosome Toxin and Immunity].—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie*. 1913. July 26. Vol. 18. No. 6. pp. 651-665.

Reference is made to the observation of SCHILLING that a suspension of nagana trypanosomes which had been allowed to stand for some hours at 37° C. is exceedingly toxic for mice and rapidly causes death. A heavily infected nagana rat was bled into 2 per cent. sodium citrate bouillon (1 part blood to 4 parts citrate bouillon); the suspension was kept at 37° C. in a water bath. The

trypanosomes, which were at first actively motile, had at the end of 3 hours become almost completely immobile. The microscopic appearances of the degenerated trypanosomes are described. Mice were inoculated intraperitoneally with .25 cc. of the suspension after it had been kept at 37° C. for the following periods, viz.,  $\frac{1}{2}$  hr., 1 hr., 3 hrs., 5 hrs. and 7 hrs. Those inoculated with the suspension which had been kept up to 3 hours became infected and died from the infection, whereas those inoculated after 5 and 7 hours respectively did not become infected, but nevertheless died. All those inoculated after 7 hours died the following night. A second series of experiments was performed in which the trypanosomes were separated from the bouillon and blood by centrifuging and then added to fresh bouillon. In this case the trypanosomes were found to die more quickly, so that at the end of one hour there was at the most only a trace of movement. Apparently the centrifugation had to a certain extent damaged the trypanosomes; the suspension was not infective after 1 $\frac{1}{2}$  hours. However, so much toxin had been formed after 2 $\frac{1}{2}$  hours that one of 2 mice died in 48 hours; after 4 $\frac{1}{2}$  hours it killed mice within 24 hours. Experiments were undertaken to show that the poison came from the trypanosomes only and not from the blood or medium. The poison was found to develop equally well when the suspension was kept at 45° C. for 4 hours, but did not develop at 56° C. Further experiments demonstrated that bacterial contamination was not responsible for the toxin formation.

The lethal dose is dependent on two factors: first the number of trypanosomes in the suspension and secondly the individual variations in the experimental animals.

The poisonous suspension was centrifuged and the deposit of disintegrated trypanosomes and the clear supernatant fluid containing the soluble constituents of the parasites were injected into different series of mice. Only those died into which the deposit was injected. When the suspension was allowed to stand at 37° C. for more than 8 hours the toxic effect began to disappear and after 18 hours it was almost completely lost. The poison is thermolabile, losing its toxicity in  $\frac{1}{2}$  hour at 56° C.; on the other hand it remains active many days when kept in the ice chest. Its action is destroyed by the addition of carbolic acid.

The poison from the trypanosomes acts as antigen. A proportion of mice treated with a non-lethal dose remained alive when subsequently, after 5-6 days, inoculated with a lethal dose sufficient to kill the control animals in 1-2 days. Doses of .0005-.001 cc. suffice to raise the resistance of the animals considerably. Further experiments were undertaken to ascertain whether the poison which had been inactivated by heating to 56° C. or by the addition of carbolic acid was still capable of acting as antigen. This proved to be the case and hence it was shown that the toxic function and the antigen function of the warmed trypanosome suspension are different.

It is of interest to observe that such antibodies are formed very early in the course of nagana infection in mice. This point is illustrated by experiments in which it was shown that mice which

were injected with infective blood + toxin did not die from the poison, but only later from the infection.

The authors were unable to demonstrate poisonous effects of suspensions from non-pathogenic trypanosomes such as *T. lewisi*. Definite results were not obtained with *T. equiperdum*, but these experiments are to be repeated. In the case of *Sp. duttoni* the authors were able to demonstrate the formation of a toxin in suspensions which had stood for 4 hours at 37° C., but doses of .25 cc. did not always kill mice within 18 hours.

It has been shown by the experiments of TEICHMANN and BRAUN (*Sleeping Sickness Bulletin*, Vol. 4, p. 58), and also by those of SCHILLING that the bodies of trypanosomes contain immuno-antigen which when introduced into a susceptible animal gives rise to a more or less marked degree of immunity against living trypanosomes. Six mice which had been treated with .25 cc. of trypanosome suspension (warmed at 37° C. for 1½-1¾ hours) were inoculated 7 days later with infective blood. The animals did not become infected. This experiment shows that immuno-antigen is present before a demonstrable amount of toxin is formed in the warmed trypanosome suspension. Another series of mice was treated with non-lethal doses (.04-.1 cc.) of the poisonous suspension and 7 days later inoculated with infective blood. Of the 9 animals thus treated 6 remained free from infection. Still better results were obtained by injecting in the first place a non-lethal dose of the poison or one which had been inactivated by heating to 56° C. and subsequently repeating the injection with a larger amount of the poison.

When the poisonous suspension was passed through a Berkefeld filter the filtrate was no longer toxic, but nevertheless served as immuno-antigen. The immuno-antigen is not destroyed by heating to 37° C. for 18 hours, whilst as already mentioned the toxin is destroyed. It was found that the carbolised poison protected against the subsequent injection of the toxin but not against the living trypanosomes.

W. Y.

AOKI (K.) & KODAMA (H.). *Beitrag zur Frage der Immunisierung mit abgetöteten Trypanosomen*. [On the Question of Immunisation with Dead Trypanosomes.]—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie*. 1913. July 26. Vol. 18. No. 6. pp. 693-700.

The question as to whether experimental animals can be immunised against trypanosome infections by dead trypanosomes is still undecided. A summary of the work which has been done along these lines is given. In their first series of experiments the authors attempted to immunise animals by means of a suspension of trypanosomes (*T. equiperdum*) which had been killed by formalin. Twelve rabbits were injected as follows:—Two animals 6 times at 6-12 day intervals with 1-2 cc. of the suspension and the other ten 3 times at 6-7 day intervals with 3 cc. Three of the animals died during the process. A week after the last vaccination all the remaining animals were inoculated with 1 cc. of virulent dourine blood; they all became

infected in a manner exactly similar to the controls. Eight rats and 17 mice were treated in a similar way with equally negative results. These experiments show that it is impossible to immunise small experimental animals by means of dead trypanosomes. As it was possible that the formalin material might not be satisfactory a further series of experiments were undertaken with dried trypanosomes. The trypanosomes were obtained from heavily infected rats by centrifugation of the citrated blood and after being once washed in normal salt solution were dried in an incubator at 37° C. for 20 hours; the mass was subsequently pulverised. Five rabbits, 10 rats and 10 mice were treated in a manner similar to that described above with the dried trypanosomes, but on subsequent inoculation with virulent blood they became infected in the ordinary way.

W. Y.

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OEHLER (R.). *Zur Gewinnung reiner Trypanosomenstämme.* [The Production of Pure Trypanosome Strains.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. July 29. Vol. 70. Nos. 1/2. pp. 110-111.

In a previous paper (see this *Bulletin*, Vol. 1, p. 525) the author showed that single trypanosomes could be separated in glass tubes and that experimental animals could be infected with them. He has carried out further experiments along these lines. A strain was carried on to the fourth passage by single trypanosome inoculation. The incubation and course of the disease remained the same. White mice injected with such a strain of *T. brucei* were treated with salvarsan and the salvarsan resistance of the strain was raised from .4 mgm. to 5 mgm. The descendants of a chosen single trypanosome exhibited salvarsan resistance to just as great an extent as the strain carried on in the ordinary way. The capacity of resistance is, therefore, a general property of all individuals of the strain. Furthermore the salvarsan-fast strain was carried on by isolation and inoculation of a single trypanosome, and the resulting infection proved just as arsenic-fast as the previous strain.

An animal was infected with an arsenic-fast and a normal strain and the author succeeded by means of inoculating single trypanosomes in isolating both components.\*

W. Y.

SCHERN (Kurt) & CITRON (Heinrich). *Ueber Lävulosurie, sowie neuartige Serum- und Leberstoffe bei Trypanosomiasis.* [On Laevulosuria as well as new Serum and Liver Substances in Trypanosomiasis.]—*Deut. Med. Wochenschr.* 1913. July 10. Vol. 39. No. 28. pp. 1356-1357.

Reference is made to the previous work of SCHERN (*Sleeping Sickness Bulletin*, Vol. 4, p. 63) wherein it was shown that Dourine trypanosomes which had become immobile on mixing

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\* It has been suggested that the formation of fast strains is due to a process of selection, some individuals of the strain having been resistant from the first (COCKERELL; LEVADITI and TWORT). Oehler's results show that this explanation will not hold, at any rate, for drug-fast strains.—A. G. B.

infected rat's blood *in vivo* with sodium citrate or normal saline solution regained their activity when fresh serum from a normal rat or horse was added. The observations recorded in the previous paper are confirmed; they were as follows:—

The length of time the trypanosomes remain motile outside the animal body depends on various factors, especially on the stage of the infection. Trypanosomes taken from an animal at the beginning of the infection may retain their motility for several hours at room temperature, whereas those obtained from an animal in the later stage of the disease, shortly before death, are much more labile and have as a rule lost their motility in 10-15 minutes. The length of time the parasites live *in vivo* is some criterion as to the probable duration of the animal's life: if the trypanosomes quickly lose their motility then it is to be expected that the death of the animal is imminent. The question arose as to whether only the serum possessed this life-prolonging property. Emulsions of liver were found to act similarly. Further experiments concerning the nature of the life-prolonging substances showed (1) that they are thermostabile, (2) that they withstand drying, (3) that they persist for a long time at 0° C. and at 37° C., and (4) that they are destroyed by putrefaction. The authors succeeded in obtaining extracts of liver and serum which contained the substance in question in concentrated form and which had been kept for four years at room temperature without losing its properties. The life-prolonging substance is not demonstrable in the serum of rats in a late stage of dourine infection, nor is it found in the fresh liver of rats which have succumbed from trypanosomiasis in the same amount as in the liver of normal animals. It was found that after treatment with atoxyl the substance—even if it had completely disappeared from the serum—gradually returned as the trypanosomes disappeared from the blood. If the animal remained cured the serum continued to be active, but if a relapse occurred the life-prolonging substance disappeared once more. It is suggested that by aid of this reaction the action of the remedy can be controlled.

Possibly these facts could be explained by a functional disturbance of the liver during trypanosome infection. Experiments were undertaken to decide the point. If animals with disease of the liver are fed with large amounts of laevulose this passes into the urine (alimentary laevulosuria); in normal animals this does not occur. As it is difficult to feed rats with a definite quantity of laevulose the sugar was administered intraperitoneally in physiological salt solution. Before proceeding with the experiments the largest amount of laevulose which could be administered to a healthy rat without the sugar appearing in the urine was determined. It was found that .3 gm. of the sugar was always completely used up in the body of large rats. This amount was then given to a large number of infected animals. The urine of these rats in the earlier and middle stages of the disease was always free from sugar. On the other hand the urine of those animals in which the trypanosomes remained motile only a short time in microscopic preparations and which were thus in the final stage of the disease, fermented on the addition of yeast. So soon as a definite reduction of the length of life of the trypanosomes of an infected rat occurs laevulose is passed in the urine after intraperitoneal injections of the sugar.

The authors write that through these experiments a new insight into the pathology of trypanosomiasis in rats is obtained. They are at present unable to state whether their experiments possess any significance for trypanosomiasis and spirillosis of

man and other animals than rats. It is also still to be demonstrated whether the diminution of the substance in question and the laevulosuria is specific for trypanosomiasis.

The conclusion is:—In rats suffering from trypanosomiasis (dourine, nagana or sleeping sickness) a functional disturbance of the liver can be demonstrated in the last stages of the infection.

W. Y.

FRY (W. B.) & RANKEN (H. S.). Further Researches on the Extrusion of Granules by Trypanosomes and on their Further Development. (With a Note on Methods by H. G. PLIMMER).—*Proc. Roy. Soc.* 1913. June 12. Vol. B. 86. No. B. 589. pp. 377-393. With 3 plates.

The methods used by the authors were (1) dark ground illumination and (2) "vital staining" with 75 per cent. toluidine blue in physiological salt solution. Methods of fixation devised by PLIMMER were followed. The trypanosomes used were *T. gambiense* (Sudan), *T. rhodesiense*, *T. brucei*, *T. evansi* (Sudan), *T. nanum* (Sudan), *T. pecaui* (Sudan), *T. lewisi*. Two classes of granules were distinguished:—(1) Infective vital granules, probably of nuclear origin; (2) others, non-infective and probably nutritive. Infective granules only were considered.

In all cases the granule, as seen by dark ground illumination, is small, sharply defined, highly refractile. With toluidine blue it stains deeper than the rest of the trypanosome body. The "sequence of events during an exacerbation" of trypanosomiasis is:—"(1) Trypanosomes without granules. (2) Trypanosomes showing granules which gradually become larger and very evident. (3) Many free granules. (4) Many trypanosomes but no contained granules. (5) Trypanolytic crisis or death of the animal." The mechanism of extrusion of granules was studied in *T. nanum* and *T. gambiense*. In the latter, the granules are multiple, move backwards and forwards in the long axis of the organism and are usually extruded from some point near the middle of the body. Extrusion of granules can be stimulated by drugs or mechanical effects, e.g. variations in osmotic conditions. Usually extrusion of granules does not seem to have a prejudicial effect on the trypanosome as seen in warm, wet preparations. "Extrusion of granules, if occurring generally, apparently heralds the disappearance of trypanosomes from the blood." Three methods by which granules may be liberated are described.

In animal infections and in human cases of sleeping sickness granules are found in the blood, glands and internal organs. Regarding further development of the granules, the authors state that "further stages are more difficult to follow, as all stages cannot be seen in any individual preparation." Elongation occurs, a Leishmania-like form being produced. "Death in acute trypanosomiasis is caused by plugging of the cerebral capillaries with these forms." Each Leishmania-like body may then elongate direct, or schizogony may occur prior to the assumption of the flagellate form. Enormous numbers of these small bodies occur in the internal organs, especially in the lungs.



Granules free from trypanosomes were obtained by mixing infected blood with hypertonic salt solution up to 2 per cent., one volume of blood with 2 or 3 of salt being used, and maintained at 34° C. to 38° C. Gerbils inoculated with such granules of *T. nanum* became infected.

PLIMMER's fixation, involving the use of iodine as a vapour or in solution, is necessary for demonstrating these granules to advantage.

[Regarding these interesting observations, a short comment may be added on the rounded forms of trypanosomes. Such were described by MOORE and BREINL (1907), and by FANTHAM (1911), the latter worker definitely comparing the rounded forms with *Leishmania*. They were considered as part of the trypanosome cycle in the vertebrate host. These views have been criticised in a somewhat unduly severe manner, apparently because LAVERAN and MESNIL in 1903 considered some rounded bodies to be "involution" forms. But FANTHAM showed that *Leishmania*-like forms of *T. gambiense* and *T. rhodesiense* were infective to experimental animals, and LAVERAN confirmed this, though still interpreting the rounded bodies as involution forms. Some trypanosomes, when degenerating, become rounded, but their nuclei show some degeneration phenomena. The mistake has been made by some of generalising that all rounded forms are the same and all are degenerate. Rounded *Leishmania*-like forms are known to be definite cyclical stages in a number of nearly allied genera of flagellates, and it now remains not only to confirm the interesting results of Fry and Ranken, but also to test their applicability to other organisms].

A. Porter.

BONGER (C.). Über die Morphologie und das Verhalten der von P. Behn in deutschen Rindern nachgewiesenen Trypanosomen bei künstlicher Infektion. [On the Morphology and the Behaviour of the Trypanosome described by P. Behn in German Cattle on Artificial Infection.]—*Zeitschr. f. Hygiene u. Infektionskr.* 1913. July 17. Vol. 75. No. 1. pp. 101-117. With 1 plate.

The paper commences with an account of the previous work on *T. theileri* and allied trypanosomes. Reference is made to the work of BEHN (*Sleeping Sickness Bulletin* Vol. 4, p. 111.).

The parasite found by BEHN in German cattle and maintained by the author by passage through calves exhibits three chief forms—long slender varieties, large broad varieties and short slender varieties. Its length is 20-70 microns and its breadth 2-6 microns. Only cattle were successfully inoculated with the parasite; sheep, goats, rabbits, guinea-pigs and mice were refractory, and as BEHN has shown not all cattle are susceptible. The average incubation period was 7 days. A febrile reaction was observed before trypanosomes appeared in the peripheral blood. The average maximum temperature of all experimental animals was 40.3° C. Beyond the temperature no symptoms were observed.

The author compares his experimental results with those of THEILER and comes to the conclusion that *T. theileri* (BRUCE and LAVERAN) and that discovered by BEHN in German cattle must, if they are not identical, be very closely related.

W. Y.

CARPANO (M.). *Trypanosomen vom Typus des Tr. Theileri in den Bindern der Kolonie Erythräa*. [Trypanosomes of the *T. theileri* Type in Cattle in Erythrea].—*Centralbl. f. Bakt.* 1. Abt. Orig. 1913. Aug. 23. Vol. 70. No. 5-6. pp. 209-217. With 1 plate.

Systematic examination of the blood of sick cattle in Erythrea revealed not uncommonly the presence of a trypanosome of the *Theileri* type. The following are the conclusions:—

In cattle of Erythrea a large trypanosome is found belonging to the *Theileri* type.

It is not found alone, but is generally associated with *Pir. bigeminum*, *Pir. mutans*, *Anaplasma marginale*, *T. abyssinicum*, *Sp. bovis* and is most frequently found in cattle sick of rinderpest.

The clinical picture varies and is dependent on the accompanying disease; not uncommonly it presents the features of Galziecte or gall fever.

The parasite can be cultivated in the NOVY-McNEAL medium (a mixture of agar with the defibrinated blood of an infected ox). In cultures the parasites acquire a peculiar morphology approximating to *Herpetomonas* and *Critidia*.

The cultures die out after about 20 days, owing to degeneration of the parasites.

Transmission through inoculation of infected blood does not always succeed in the cattle of Erythrea. The inoculated cattle must be susceptible for the parasites which in general accompany this trypanosome, especially *Piroplasma bigeminum*.

W. Y.

RONDONI (P.) & GORETTI (G.). *Ricerche Sperimentali sul Nagana*.  
i. *Comunicazione. Su alcune Proprietà Biologiche della Milza nella Infesione Sperimentale da Trypanosoma brucei*. [Experimental Researches on Nagana].—*Lo Sperimentale*. 1913. April 7. Vol. 67. No. 1. pp. 1-24.

The authors of this paper give an account of experiments which they undertook in order to study certain properties of the spleen of guinea-pigs, rats and mice during the course of an infection with *Trypanosoma brucei*. They endeavoured to demonstrate the presence of trypanocidal substances and specific antigens in the spleen, and also studied its haemolytic powers.

A description of the technique adopted for each portion of their experiments is given in detail.

In the spleen there is sometimes a certain degree of special trypanolytic power; the spleen must have a certain importance in the defence of the organism, it is something more than a simple filter in which accumulate fragments of trypanosomes already dead. However, it appears not to have in it a marked accumulation of trypanosomal antigens such as one might use, for example, for experiments and attempts at immunisation.

Aqueous extracts of the spleen of infected animals possess a definite haemolytic power on erythrocytes of whatever origin

(even from the same individual); two active substances can be extracted by alcohol; they behave irregularly as regards heat; they are capable of being inhibited, in part at least, by normal serum; they are probably of lipoid nature and have but slight importance during life.

W. Y.

ARAGAO (Henrique de Beaurepaire). **Nota sobre as Schizogonias e Gametogonias dos Trypanosomos.**—*Brazil Medico*. 1913. July 15. Vol. 27. No. 27. pp. 271-272.

The paper consists of a review of the results obtained by various workers on the schizogony of different trypanosomes, following the discovery of that process in *Trypanosoma cruzi* by CHAGAS, together with criticisms of the same.

Aragao professes a certain amount of scepticism with regard to the connection of the pulmonary cysts, usually considered as multiplicative stages of *T. cruzi*, and the flagellate of Brazilian trypanosomiasis. In his experiments, schizogonic cysts occurred in normal animals; 8 out of 31 dogs, 11 out of 46 guinea-pigs and 2 out of 7 rats containing them. This was confirmed by DELANOË who also found them in other animals in which no trypanosomes, as such, were present. DELANOË thinks that the pneumocysts may belong to the Coccidia, while TIZZER thinks they are Cryptosporidia. In some animals, pneumocysts appear to produce no pathological alterations and seem to be inoffensive to the host.

A. P.

STRACHAN (Henry). **West African Notes.** (1) **Note on a Portable Insect-proof Room.** (2) **Notes on the Bites of the Tsetse-Fly (*Glossina palpalis*).**—*Jl. Trop. Med. & Hyg.* 1913. July 15. Vol. 16. No. 14. p. 214.

1. Note on a portable insect-proof room, constructed by Messrs. Conner and Sons, 263, Lewisham High Road, S.E.

2. Notes on the bites of *Glossina palpalis*. The attack of the fly varied strangely. It was sometimes accompanied by a loud buzz and violent impact, with a sharp and painful stab. At others it was silent and painless, and it not unfrequently happened that only the discovery that blood was staining the site of the puncture revealed the fact that one had been bitten. The author is of opinion that there is grave danger of sleeping sickness spreading southward along the river valley into the western province of Southern Nigeria.

W. Y.

## RELAPSING FEVER.

MARCHOUX (E.) & COUVY (L.). *Argas et Spirochètes*. (Premier Mémoire.)—*Ann. Inst. Pasteur*. 1913. June. Vol. 27. No. 6. pp. 450-480. With 15 text-figs.

This first part of this interesting memoir is devoted to a historical account of LEISHMAN'S granules, first observed by DUTTON and TODD in the Malphigian tubules of *O. moubata* infected with *S. duttoni*, and subsequently by LEISHMAN, BALFOUR, HINDLE, and FANTHAM. The authors then proceed to discuss the origin of the granules, which are stated to be derived from the breaking up of little masses of protoplasm occurring in the cells of the Malphigian tubules, ovules, and the genital canals of the male and female.

When fully formed, the granules are ovoid bodies about  $0.5 \times 1\mu$  and are agglomerated into large masses simulating schizogony. In the Malphigian tubules they may become so numerous as to completely fill the cytoplasm of the cells, but in the ovules and cells of the genital ducts the granules are less numerous, occurring in little groups, or sometimes singly. The shape of the granules is not constant, for, especially in the genital ducts, they are sometimes bacilliform.

The authors then consider the question of the relation of the granules to the spirochaetes. In the first place the disappearance of spirochaetes from the tick is more apparent than real. (See this *Bulletin*, Vol. 1, p. 395.) By staining with gentian violet it is possible to detect spirochaetes in the coelomic fluid of ticks submitted to the following processes:—(1) Starvation for forty-five days at  $28^{\circ}\text{C}.$ , and (2) for eleven months at  $15^{\circ}\text{C}.$  In the latter case attenuated spirochaetes could still be found in small numbers in the coelomic fluid, and more or less normal spirochaetes in the salivary ducts and the interior of the palps. (3) Five months starvation, the ticks being kept alternately in the ice-chest and the laboratory.

In addition, attempts were made to cause the disappearance of spirochaetes from the body of the tick, by feeding it on a fowl hyperimmunised against *S. gallinarum*, but this procedure was without effect. The direct inoculation of hyperimmune serum into the body cavity of *Argas* caused the spirochaetes to disappear for the moment, but in three-fifths of the cases the parasites reappeared after a few days.

The thickness of the spirochaetes diminishes when the ticks are starved, but the normal dimensions are recovered after the tick has ingested blood. Employing \*HINDLE'S method certain *Argas* were fed on normal saline, after which it was found that the attenuated spirochaetes were quite unaffected in thickness. Moreover, some ticks were fed on blood and others, of the same lot, on normal saline. Spirochaetes were excessively rare and

\* HINDLE & MERRIMAN. The Sensory Perceptions of *Argas persicus* (Oken).—*Parasitology*. 1912. Vol. 5. pp. 203-216.

were extremely slender in the coelomic fluid of the latter, whilst as many as ten normal spirochaetes could be found in a drop of coelomic fluid from those ticks that fed on blood. These attenuated spirochaetes are said to be virulent, for when injected into fowls they produced infection.

The authors were able to observe the rapid passage of the spirochaetes from the gut through the gut wall into the coelomic fluid, thus confirming HINDLE'S observations. Certain of the spirochaetes, however, remain in the lumen of the gut, disappearing eight to twelve days later. During this period the spirochaetes break up into granules, and also short spirilla may be found, but these are to be regarded as merely degeneration forms.

With regard to the nature of LEISHMAN'S granules the authors are unable to give a definite opinion but bring forward evidence to show that they are not a stage in the development of the spirochaete. When the bodies are maintained at a temperature of about 37° C., they were observed to elongate and assume bacilloid and spirilloid forms, but without ever becoming transformed into normal spirochaetes. Moreover the granules may be found in the Malphigian tubules of *Argas respertilionis*, *Rhipicephalus sanguineus*, *Haemaphysalis concinna*, *Amblyomma variegatum*, *Dermacentor reticulatus*, *Ixodes ricinus*, and *Lelaps echidninus*.

The authors' conclusions are as follows:—

(1) In *Argas persicus* granules are present similar to those described for the first time by LEISHMAN in *O. moubata*.

(2) These granules have no relation to the spirochaetes.

(3) It is impossible to inoculate them into an animal without at the same time inoculating spirochaetes.

(4) We have found spirochaetes in the coelom of all the *A. persicus* that we have examined.

(5) These spirochaetes are often so fine that they are not colourable by Giemsa, but may be put in evidence by staining with gentian violet.

(6) There are other spirochaetes so fine that they remain invisible whatever the staining method employed.

(7) We have not succeeded in obtaining *Argas* free from spirochaetes.

(8) A large number of the spirochaetes ingested by the *Argas* almost immediately pass through the wall of the alimentary canal and appear in the coelomic fluid.

(9) Those which remain in the gut degenerate in eight to twelve days.

(10) The fragmentation of the chromatin in the spirochaetes must not be considered as a transformation of these organisms into granules, but as a process of degeneration.

(11) Leishman's granules differ from spirochaetes in a large number of characters and reactions.

(12) They exist normally in numerous acarions.

(13) We have not observed any changes in the spirochaetes whilst in the invertebrate host, permitting one to cease from regarding these parasites as bacteria.

[This important memoir should be read in its entirety by those interested in the nature of spirochaetes and their life-cycle within their respective hosts. The evidence brought forward by the authors seems to suggest that the granules have no relation to spirochaetes.]

E. Hindle.

**MACKIE (F. P.).** The Body-louse (*Pediculus vestimenti*) as a Disease Carrier. The Body-louse as a Carrier of Relapsing Fever.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 281-289. (1913. Simla: Govt. Central Branch Press.)

The present article is an interesting summary of our knowledge of the body-louse as a disease carrier and is divided into three parts, the first of which is concerned with the body-louse as a carrier of relapsing fever, the second with the body-louse as a carrier of typhus fever, and a third part with the bionomics of the body-louse. In the first part the author recalls his observations made during the Nasik epidemic of Indian relapsing fever (*S. carteri*) in August, 1907.\* It may be noted that this outbreak occurred in a mission settlement amongst a juvenile population. It broke out in the boys' ward and in about one month 137 out of 145 boys were struck down. Later it spread to the girls' ward but there it only progressed very slowly and ultimately only 35 out of 115 girls became infected. The boys were swarming with body-lice whilst these parasites were much less common on the girls; on the other hand, bugs were very abundant in the girls' ward but were absent from the boys' quarters. On dissecting the lice obtained in the wards during the height of the epidemic, out of 112 from the boys' ward, 24.1 per cent. were infected with spirilla whilst out of 29 obtained from the girls' ward only one was infected.

The chief seat of the multiplication of the spirochaetes appears to be the alimentary canal, and Mackie thinks it possible that the infection travels forward to the pharynx and mouth-parts of the louse. About 100 eggs were examined but no spirilla found.

The remainder of this section and the next, dealing with the body-louse and typhus fever, consist of a summary of the more important literature on the subject.

In the section dealing with the bionomics of the body-louse the author gives short notes on the anatomy and breeding habits of these insects. In India both eggs and adults do best in a cool incubator. The length of life of the adults was found to be only about 7-10 days, but the insects were only fed once a day and WARBURTON† states that it is necessary to feed them twice a day in order to keep them in good health.

Over 100 head-lice from infected boys and girls were examined at Nasik, but no blood was ever found in the insects, their only food seeming to be a greasy sebaceous material. It is noted, however, that PATTON found blood in head lice.

\* **MACKIE:** The Part played by *Pediculus corporis* in the Transmission of Relapsing Fever.—*Brit. Med. Jl.* 1907. Dec. 14. pp. 1706-1709.

† **WARBURTON:** A Preliminary Investigation on Flock as a possible Distributor of Vermin, and on the Life History of the Body-louse (*Pediculus vestimenti*).—*Repts. to the Local Govt. Board on Public Health & Medical Subjects.* 1909. New Series No. 2. p. 3; also 1910. New Series No. 27. p. 25.

The author recalls that he first observed the flagellate occurring in the body-louse and subsequently described by FANTHAM as *Herpetomonas vestimenti*. The presence of this flagellate must be remembered by those investigating the disease carrying power of the louse, especially in kala azar. In Madras last May the author fed 279 body-lice on patients suffering from this disease who had *Leishmania donovani* in their peripheral blood. On subsequently dissecting the lice at different intervals no flagellates of any sort could be found.

Finally it is pointed out that the body-louse is one of the easiest of all blood-sucking parasites to eradicate, for personal cleanliness and the disinfection or even plain washing of clothes is sufficient to kill the lice, especially if the garments are afterwards exposed to the sun to dry. It is probable that the isolation of patients together with the disinfection of clothes and bedding, and the avoidance of actual contact, will suffice to cut short epidemics of relapsing fever and typhus.

[Without wishing in any way to detract from the credit due to Captain Mackie for his important observations, his opening statement that "the first record of the louse being suspected as a carrier of a specific disease dates from the publication of the observations made at Nasik in August 1907 on an epidemic of Relapsing Fever," is not strictly accurate. As far back as 1891 FLÜGGE\* suggested that lice were responsible for the transmission of European Relapsing Fever. Moreover, with regard to other infections DEWÈVRE (1892)† performed experiments proving that impetigo was carried by lice, whilst AUBERT (1879)‡ considered that these parasites caused impetigo, prurigo, pityriasis, etc., and especially favus.]

E. H.

TODD (John L.). **A Note on the Transmission of Spirochaetes.**—*Proc. Soc. for Experim. Biology & Med.* 1913. April. Vol. 10. No. 4. pp. 134-135. [87 (783).]

On several occasions the author has examined the coxal and anal fluids excreted by infected *Ornithodoros moubata* coming from Uganda and British Central Africa. In every instance the fluid was taken while the ticks fed upon an uninfected animal. The fluid collected was free from blood and in two instances coxal fluid was obtained free from anal secretion. On six occasions, after the fluid had been centrifuged, spirochaetes morphologically identical with *S. duttoni* were found in it. In addition spirochaetes were found in the fluid that apparently contained no anal secretion.

[Dr. Todd's observation is the first record of spirochaetes, as such, being found in the coxal fluid and anal secretions and is

\* FLÜGGE C. (1891) *Grundriss der Hygiene*. pp. 473 and 532.

† DEWÈVRE (1892) Note sur le Rôle des Pediculi dans la Propagation de l'Impetigo.—*Compt. Rend. Soc. Biol. Paris*. 1892. No. 11. pp. 252-254.

‡ AUBERT (1879) Les Poux et les Ecoles. Un Point d'Hygiène Scolaire. Lyon, 1879. Reviewed in *Ann. de Dermatologie et de Syphiligraphie*, 1880. 2 ser. vol. 1. p. 292.

of considerable interest in view of LEISHMAN's suggestion that, when a tick feeds, the infection is produced by the entrance of these fluids into the open wound caused by the tick's bite.]

E. H.

JUKES (A. M.). *Spirillar Fever in the Darjeeling District, 1912.*—*Indian Med. Gaz.* 1913. June. Vol. 48. No. 6. pp. 222-225.

The present article contains a more detailed account of a spirillar fever occurring in the Darjeeling District, which was previously described by the author (see this *Bulletin*, Vol. 1, pp. 387-388). In every case the illness is severe. The temperature is irregular and shows no resemblance to that of relapsing fever, and no relapses have been observed in the case of those that recovered. The author is inclined to regard the fever as hitherto undescribed, and adds some notes by Capt. MACKIE on the morphology of the spirochaete causing the disease. The parasites tend to group themselves into two ranges, the shorter from  $9.11\mu$ , and the larger from  $15.18\mu$  in length, and this is what often happens in the Bombay form. On the whole, however, they appear to be somewhat shorter than the type specimens of *S. carteri*, but this is not a very reliable guide. Lice and bugs from infected houses were dissected, but in no cases were spirochaetes found in any of these insects.

The author adds some further observations by Sub-Assistant Surgeon SWOLE who at Kurseong in June saw five cases of similar fever with two deaths in people who had recently come from Tibet. Spirochaetes were found in the blood of one of them, so it seems probable that this fever also occurs in Tibet. In addition the coolies of the Takvar Estate stated that the fever is well-known in Nepaul and that it is very fatal, but if a patient lives till the eighth day he generally recovers. The author heard on reliable information that in another epidemic on a tea-garden, twenty out of twenty-one persons died. The disease was still continuing in December. In the appendix detailed symptoms of three cases are given.

E. H.

RIZZUTI (G.). *Osservazioni sul Tifo Ricorrente a Tripoli.* [Observations on Relapsing Fever in Tripoli.]—*Malaria e Malat. d. Paesi Caldi.* 1913. April-May. Vol. 4. No. 3. pp. 153-172.

During the first period of the Italian military occupation of Libya, from December 1911 to March 1912, the author observed 25 cases of relapsing fever among the soldiers. In all cases the clinical symptoms resembled those described for the relapsing fevers of other parts of North Africa, but were rather mild, for none of the patients succumbed to the disease, though no medicaments were employed.



Detailed descriptions are given of the clinical symptoms of the disease in sixteen of the patients, but none of them call for any special notice. Icterus was noticed in ten per cent., vomiting in 50 per cent., epistaxis in 25 per cent., haematuria in three per cent., splenomegaly in 80 per cent., and enlarged liver in 15 per cent. of the cases. During the fever there was a decrease in the percentage of haemaglobin, and the number of "Rossi's corpuscles," accompanied by a well marked increase in the number of leucocytes. The spirochaetes were never very numerous in the blood, not more than 1-2 per microscopic field, and agglutinated masses of the parasites were never observed. In defibrinated blood at ordinary temperature the spirochaetes remained motile for seven days; and in hanging drop preparations for three to four days. Mice were infected by the subcutaneous injection of 1 cc. of the blood from an infected patient, but the results of the experimental work with animals are reserved for a future communication.

The author adds that all the cases of relapsing fever, as well as those of typhus (some of which also occurred amongst the troops), agree with the view that lice are responsible for the transmission of the infections.

E. H.

- KUSUNOKI (F.). Experimentelle Untersuchungen über Heredo-Immunität bei afrikanischer Recurrens und über den etwaigen Einfluss von Immunitätsvorgängen auf die Wirksamkeit eines chemotherapeutischen Mittels. [An Experimental Investigation of the Inheritance of Immunity in African Relapsing Fever and the Possible Influence of Immunity on the Action of Chemotherapeutic Substances.]—*Zeitschr. f. Chemotherapie*. 1. Abt., Orig. 1913. Vol. 2. No. 1. pp. 11-22.

Working with a very virulent strain of *S. duttoni*, the author found that rats and mice that had recovered from an infection could not be reinfected after an interval of two months. Young rats born whilst the mother was infected or after she had recovered from an infection were found to be quite susceptible when inoculated subcutaneously with the same virus. It seems, therefore, that in the case of infections of *S. duttoni* no immunity is transmitted to the offspring.

In order to test the possible influence of immune substances on the action of salvarsan the author inoculated fifteen mice with *S. duttoni*. Five of these were used as controls and all died; six were injected on the second day with 1 cc. of a 1/900 solution of salvarsan per 20 mgm. body weight, whilst of the remaining four mice, two were injected with the same dose on the fourth day of the infection and two on the sixth day. Three of those treated on the second day of infection recovered, whilst all the others succumbed, so that the presence of immune substances does not seem to influence the action of the drug. It should be noted that, according to HATA, the smallest curative dose for

*S. duttoni* in mice is 1 cc. of a 1/700 to 1/800 solution of salvarsan per 20 gm. body weight and the author purposely used a smaller dose in order to detect whether the presence of immune substances had any effect. The results of this and also two other experiments, in which the salvarsan was administered prior to the infection, show that in the case of infections of *S. duttoni* the action of this compound is not in any way affected by development of any natural immunity in the host.

E. H.

CHAMBERS (Helen). **A New Spirochaeta found in Human Blood. Preliminary Communication.**—*Lancet*. 1913. June 21. pp. 1728-1729. With 5 text-figs.

The author claims to have found spirochaetes in the blood of a number of persons both healthy and unhealthy. A total of 47 cases was examined and the organism detected in all except three.

The blood was collected in small Wright's tubes and allowed to clot at 37° C. for twenty minutes. The serum was then pipetted off and the remainder of the clot returned to the incubator; after 1½ hours the serum which separated, owing to further shrinkage, was examined by dark-ground illumination. The organisms are said to escape into the serum chiefly during the last stage of contraction of the clot.

The organism is said to be variable both in length and thickness, to multiply by longitudinal division and to frequently show round spore-like bodies attached to the ends or laterally. With Giemsa and Leishman's stain small red chromatic granules are often found at the end and sometimes along the length of the body; the rest of the spirochaete stains faintly blue.

Various attempts to cultivate the organism are also described, but although it multiplies in the blood itself or in the first cultures, no subcultures could be obtained. "A broth tube containing these organisms was heated to 50° C. for an hour. They were still actively motile." (!)

[The appearances figured by Dr. Chambers have merely a very superficial resemblance to spirochaetes and from the description there can hardly be any doubt that the author has been misled by the very extraordinary appearances assumed by degenerating and coagulated blood.]

E. H.

SANGIORGI (Giuseppe). **Spirochetosi della Cavia.** [Spirochaetosis of the Guinea-pig.]—*Pathologica*. 1913. July 15. Vol. 5. No. 113. pp. 428-430. With 3 text-figs.

Whilst performing the autopsies of numerous guinea-pigs that had been infected with "Guinea-pig plague," the author on one occasion discovered a spirochaete which is apparently pathogenic. The parasite was found inhabiting the cavities of various lesions

of the liver and seemed to be mainly responsible for certain inflammatory and degenerative changes that had taken place. The spirochaetes belonged to two types, the first of which measured  $3.2\mu$  to  $4.8\mu$  in length by  $0.3\mu$  to  $0.4\mu$  in breadth, whilst the larger forms were  $9.6\mu$  to  $11.2\mu$  in length and about the same breadth as in the other type. The relations between these two types could not be decided, and also the author did not observe any traces of division. Attempts at subinoculations failed, since the guinea-pig infected with spirochaetosis had died as a result of the Guinea-pig plague, and therefore all animals inoculated from it died as a result of the latter disease before any spirochaetes could be detected.

This spirochaete, for which the name *Spiroschaudinnia caviae* is proposed, seems to be identical with that also observed in the guinea-pig by DE GASPERI. [See this *Bulletin*, Vol. 1, p. 396.]

E. H.

## TROPICAL DISEASES BUREAU

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## CHOLERA.

**SANTOLIVIDO.** *Les Administrations Sanitaires dans la Lutte contre le Choléra.* (Conférence faite à la Société Impériale de Médecine de Constantinople.)—*Bull. Office Internat. d'Hyg. Publique.* 1913. June. Vol. 5. No. 6. pp. 969-979.

Santolivido discusses the prophylactic measures to be carried out in an infected country for its own safety and to protect uninfected countries. He dwells on the fact that for practical purposes cholera is carried exclusively by man. He believes that genuine cholera nearly always precedes official cholera. He shows how it is impossible by any practicable precautionary measures at the frontiers to prevent the entrance of cholera, and how difficult it is after the disease has gained entrance to trace the point of entrance. The real cause of this difficulty is the healthy carrier. It would be quite impossible to institute a satisfactory search for carriers in a ship free from the disease or in the trains crossing the various frontiers perpetually. While these healthy carriers are a source of danger, they are not highly dangerous and strict supervision at the frontiers by detecting actual cases is an invaluable procedure.

The duties devolving on the sanitary authorities of infected countries as laid down by the last international sanitary convention are discussed. He agrees that ordinary merchandise as such has not been shown to spread cholera, but of course all linen and other personal effects of patients must be disinfected. The part played by different foods in carrying infection is discussed at considerable length and Santolivido gives cases to show that the sending of food supplies from infected to uninfected countries is, as far as is known, permissible.

The author in dealing with prophylaxis in an infected country states "He may take cholera who will." This he illustrates by an outbreak of cholera in an asylum in Italy where tying the hands of the patients and confining them all to bed, with untying of the hands and disinfecting them before meals was followed by an immediate cessation of the epidemic.

Given pure water and food, personal cleanliness and disinfection of the hands before meals mean personal safety. As a practical example of what ought to be done in checking cholera locally he gives the procedure he adopted at Naples. The treatment of patient, personal effects, house, neighbouring houses, search for carriers around patient and in all shops selling foods of different sorts, strict supervision of hotel and lodging houses, are detailed and Santoliquido expresses the conviction that such methods guarantee success.

The protection of the country locally is the best protection possible for neighbouring countries, but in addition the prevention of patients leaving the country is a duty of great importance and was effectively carried out by Italy in the late epidemic. A short reference to the anti-cholera activity of Turkey completes the paper.

W. J. Penfold.

POTTEVIN (Henri). *Les Bases Scientifiques de la Lutte contre le Choléra. (Conférence faite à la Société Impériale de Médecine de Constantinople.)—Bull. Office Internat. d'Hyg. Publique.* 1913. June. Vol. 5. No. 6. pp. 953-968.

After a few introductory remarks on the diagnosis of the disease, its seasonal prevalence, and the virulence of the virus as judged by the incidence and mortality of the disease in India, the author proceeds to discuss means of prophylaxis under three heads:—Those applying to man, water, and other possible vehicles of infection.

In the case of man the first essential is to identify those who carry the infection. This is done by clinical examination supported by bacteriological examination in doubtful cases, and in the search for healthy carriers. The routine bacteriological method advised by Pottevin consists in making 1st stained and hanging drop preparations of a suitable flake of the faeces; 2nd, plating out similar flakes on alkaline agar and on Dieudonné's medium; 3rd, adding 1 cc. of suspected material to 50 cc. of peptone water and allowing that to grow at 37°C. for six hours and then plating out. Colonies on these various plates are examined and vibrios tested for agglutination in hanging drop. Suspected vibrios are then isolated in pure culture and tested completely by agglutination and Pfeiffer's reaction.

The agglutination reaction by patients' serum is not of great practical value as it appears late in the history of the case.

The morphology of the *V. cholerae* is discussed briefly by Pottevin without anything new being added to this subject. The absolute test he employs to determine whether the organism is a genuine cholera strain or not is the power to agglutinate to 1000 with a specific serum which has a titre of not less than 4000 tested against the specific organism used to produce it.

The Pfeiffer reaction is unnecessary in practice since no evidence exists of a vibrio which agglutinates in a high degree with a specific serum, not responding to Pfeiffer's test.

In cases clinically like cholera where in spite of repeated examination the vibrio cannot be found it is undoubtedly best to

act as if they were definite cases. In children the difficulty of cholera-diagnosis makes Pottevin suggest that in all cases of gastro-enteritis, or meningitis in the young, a complete bacteriological examination ought to be made.

The joint occurrence of dysentery and cholera in the same patient tends to alter materially the clinical picture. Cramps are often absent as are also rice-watery stools, and of course marked depression of temperature is not infrequent in dysentery. It has further been shown at Tor that cholera carriers are relatively frequent amongst the dysentery patients.

The duration of the cholera-carrier state in convalescents was worked out in more than 3,000 cases in Italy, in the years 1910 and 1911, when it was found that 97 per cent. were free at the end of a month. It appears highly exceptional to carry the germ for longer than two months.

Intermittency of the carrier-state appears to occur, but Pottevin believes three successive negative examinations made at two day intervals may be taken as evidence of complete cessation. The proportion of healthy carriers found in the entourage of patients varies greatly according to results published in different countries, from 22 per 100 patients in Germany to 117 in Roumania. The search on frontiers for carriers amongst healthy people who have come from an infected country has never given a high percentage of positives, and it is only recommended by Pottevin in the case of those travellers who have been in actual contact with a cholera case on the journey.

Those measures directed to the prevention of cholera patients leaving their respective countries and the strict supervision during the journey of large masses of people leaving an infected country are insisted upon by Pottevin as essential in cholera prophylaxis.

Having found the infected and searched all the surrounding persons for carriers, suitable isolation is practised and the usual methods of disinfection. River or sea water is only a temporary support for the virus. Pottevin insists on the need for a water supply above suspicion. He looks upon as useless any guarantee that bacteriology can give of purity of water in the face of a cholera epidemic. He suggests the sterilization of doubtful water by six hours' contact with hypochlorite of soda; 1 mgm. per litre is an efficient quantity and does not give an unpleasant taste to the water.

Of other possible agents of infection articles of diet are considered. Certain foods may act as good culture media and such foods require strict supervision. The prevention of carriers handling food is obviously important.

That the danger of food carriage of infection is easily over-estimated is suggested by much practical experience, *e.g.*, the free passage of fruit between South Italy on the one hand and Central Italy, Western Italy and Switzerland on the other, never occasioned a single case of cholera though South Italy was severely infected.

Personal belongings must be treated as in the case of other infectious diseases.

W. J. P.

POTTEVIN. Contribution à l'Étiologie du Choléra.—*Bull. Office Internat. d'Hyg. Publique*. 1913. July. Vol. 5. No. 7. pp. 1158-1174.

Observations made at Tor on the carrier question in cholera constitute the first portion of the report. In the winter 1912-1913 over 14,000 samples of faeces were examined, mainly derived from Egyptian pilgrims. 7.4 per thousand showed vibrios and in 69 cases they were cholera vibrios. 1.7 per thousand of healthy carriers of cholera germs were found in over 13,000 pilgrims examined. The figure is higher than that obtained in previous work on the subject. The pilgrims could be sharply divided, according to the Egyptian route they took, into two groups, one of which consisted almost entirely of poor and decrepit people while the others were much better off. The carriers were much more numerous in the former group.

*Sick carriers*, i.e., those carrying the specific germ but sick of some malady other than cholera were frequent, reaching from 79 to 110 per thousand. The dysenteric patients especially are frequent carriers.

*Study of the Vibrios isolated*.—The total number was 127. The following tests were applied to them. (1) Liquefaction of gelatine; (2) cholera-red reaction; (3) agglutination; (4) Pfeiffer's reaction; (5) deviation of complement.

All the vibrios which were specifically agglutinable, liquefied gelatine, some very quickly and others very slightly after a week. 84 per cent. of the agglutinable vibrios gave a positive cholera-red reaction, and it appears that this reaction changes in the case of the same strain easily; negative and positive results with vibrios from the same patient do not appear to be rare.

In respect of agglutination, it was found that strains agglutinating highly on isolation, frequently lost the power to do so, and on the other hand non-agglutinating strains took on the agglutinating power almost as suddenly. Both agglutinating and non-agglutinating varieties were found simultaneously in the same intestine.

Pfeiffer's reaction was always positive with the agglutinating strains and occasionally with strains that did not agglutinate.

Deviation of complement was more or less complete in the case of all agglutinating strains but it was also complete in some non-agglutinating vibrios. 19.5 per cent. of the agglutinating strains were haemolytic.

The second portion of the paper deals with the nature of cholera toxins and antitoxins and gives similar results to those recorded in another abstract of the same author's work appearing in this number.

"The effects of the antitoxin on a few clinical cases are also mentioned and Pottevin believes it to have been of use in some. The doses used were enormous, one patient receiving 580 cc. of serum.

CHAZARAIN-WETZEL. **Accès Pernicieux Cholériforme.** [A Pernicious Cholera-like Attack.]—*Bull. Soc. Méd.-Chirurg de l'Indochine.* 1913. July. Vol. 4. No. 7. pp. 324-326.

The case was that of a European of 45 who gave a history of two previous attacks of cholera. This attack occurred in the middle of the summer. The diarrhoea was very severe and muscular cramps occurred the first day. On examination his temperature was 35° C., he was covered with a cold, clammy sweat, his voice was normal, extremities cold, pulse feeble, face cadaveric, eyes fixed and staring. He had the feeling of having an enormous weight in the pit of the stomach, a pause occurred between inspiration and expiration, urine was suppressed, evacuations were serous with greyish flocculi and of a very faecal odour, the tongue was coated and patient thirsty. Vomiting was entirely absent.

He was treated symptomatically and in the evening of the same day showed marked improvement and in a few days became entirely well. The case was brought forward for differential diagnosis; at first sight it looked like cholera but the normal voice and absence of vomiting and the nature of the motions were against that.

The author suggests that it may have been an infection with some member of the food poisoning group of organisms but the surrounding circumstances did not favour this; he prefers the view that it was a cholera-like attack, peculiar to the locality, of which the cause does not yet appear to be ascertained.

W. J. P.

GOÉRE (J.). **Le Choléra à Ferryville (Tunisie) en 1911. Etude Clinique et Bactériologique.**—*Arch. de Méd. et Pharm. Navales.* 1913. July. Vol. 100. No. 7. pp. 52-60; and Aug. No. 8. pp. 124-137.

The water locally is very bad having always at least 1,000 to 1,200 colon bacilli per litre. The naval medical authorities elaborated a precautionary scheme of defence against the disease. Their first requirement was that all water for consumption should be boiled. After ten days' experience of this it was given up as the water was objectionable to the personnel and the method was also difficult to carry out. As a substitute, water, after bacteriological examination had failed to find the *V. cholera*, was used, until cholera cases commenced to be sent to hospital, after which boiling of the water was again resorted to.

The examination of the water for the *V. cholera* was carried out by METSCHNIKOFF's method, by means of which it can be detected the next day after the laboratory receives the sample.

Attempts to sterilize the water with ultraviolet rays are described but the local water was too muddy for this method to be effective. An account of the order of occurrence of cases is given. The first was reported on September 25th. A native who was flying from an infected district was found infected with cholera, he was lying on the ground soiled with dejections, and cold. A tent was placed over him and he was treated on the spot; he died



on the eighth day after. Tuberculosis and typhoid pavilions of the local hospital were then used to accommodate the cases which occurred in a straggling fashion from time to time. The diagnosis in all cases was confirmed by bacteriological examination. The cases were promptly isolated as also a number of healthy carriers. In all 16 cases occurred at Ferryville; 8 of these were in hospital; 9 died of which only one was a hospital case. Seven healthy carriers were discovered.

Goéré describes a series of five cases. The first case was that of a sea-going engineer who had for some time before been treated for jaundice and obstinate constipation. The case was typical and the description vivid but contains nothing new. The second case was mild, it commenced 21st October and temperature was never below 36.4° C. It was treated by anticholera serum from the Pasteur Institute, 100 cc. being given. The agglutination test was not positive on the 25th or 28th October, but was positive on the 8th November tested against the vibrio obtained from the patient himself. The other three clinical histories given present nothing very unusual except the administration of very large and frequent doses of serum in treatment, which appeared to be useful.

W. J. P.

**ROELFSEMA (F. H.). Enkele korte Opmerkingen over Verloop en Behandeling van de Cholera in het Militair Hospital te Semarang in de Jaren 1910-1912.** [Short Observations on the Course and Treatment of Cholera in the Military Hospital of Semarang in the years 1910-1912.]—*Geneeskundig Tijdschr. v. Nederl.-Indië*. 1913. Vol. 53. No. 3. pp. 446-448.

The numbers treated during the three years were small, in the third year only 24. The mortality rate fell from 60 per cent. in 1910 to 25 per cent. in 1912. This appears to have been due to diminution in the virulence of the organism. The author found injection of hypertonic saline useful in treatment.

W. J. P.

**FREISE (W.). Die Epidemiologie der asiatischen Cholera seit 1899 (VI. Pandemie).** [The Epidemiology of Asiatic Cholera since 1899. The 6th Pandemic.]—*Beihefte z. Arch. f. Schiff- u. Tropen-Hyg.* 1913. June. Vol. 17. Beiheft 5. 81 pp. [pp. 289-365.] With tables, maps, and 1 curve.

This monograph is a summary of the distribution of cholera during each year from 1899 to 1911. The method of treatment of the subject is to give a list of the literature quoted at the commencement of the section dealing with the disease during the year in review and then, starting from India, to trace the spread to all the neighbouring countries and so throughout the old world. This is done for each year as far as possible from official records. Then follow synoptic tables showing at a glance the countries affected each year. Further tables are given showing the course of the epidemics in relationship to season and province in different countries, and also the effect of the pilgrims on the cholera death rate in Mecca.

W. J. P.

## LABORATORY AND EXPERIMENTAL.

SGALITZER (Max) & Löwy (Otto). Ueber die Verwendbarkeit der Blutalkalibouillon als Anreicherungsmedium für Choleravibrien. [On the Use of Alkaline Blood-broth as an enriching Medium for the *Vibrio cholerae*.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. July 3. Vol. 69. No. 7. pp. 556-560.

The authors pass in review peptone water, alkaline ox bile, and alkaline blood-peptone water as liquid enriching media for the *V. cholerae* and conclude that they are all inferior to KRAUS's alkaline blood broth. Opinions on the latter vary, but that appears to be due to the fact that the medium, though made up in the same way, is different in different batches; each batch must be tested as to its individual suitability. If this be done the authors believe it to be the best fluid enriching medium for *Vibrio cholerae*.

They detail a series of experiments to show the effect of adding to 100 cc. of neutral litmus broth different quantities of Kraus's blood-alkali, on the growth of a large number of different bacteria occurring in faeces. They found in the particular sample they used that if the enriching culture were to grow six hours before plating then 20 cc. was the best quantity to add to get the most specific effect.

W. J. P.

KABESHIMA (T.). Ueber einen Hämoglobineextrakt-Soda-Agar als Elektivnährboden für Choleravibrien. [On an Alkaline Haemoglobin-Extract-Agar as a selective Medium in Cholera Diagnosis.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Aug. 4. Vol. 70. No. 3-4. pp. 202-208.

Kabeshima discusses first of all the disadvantages of the special cholera-agar media of DIEUDONNÉ, ESCH and PILON. In his research he set himself the task of producing a suitable agar for the plating out of cholera stools which could be used quickly, made easily, and would also suppress effectively non-cholera vibrios as well as the members of the typhoid-coli group.

For this purpose he suggests as the result of his work an alkaline agar to which Pfeiffer's haemoglobin extract in suitable quantity is added. He tested this agar against the above-mentioned agars as controls in the case of a large number of laboratory strains of cholera, other vibrios and various members of the typhoid-coli group and also in the case of the stools from 21 patients suffering from diarrhoea, which stools he artificially infected with *V. cholerae*.

These experiments showed that Kabeshima's agar was an excellent medium for all the cholera strains, it restrained some non-cholera vibrios, and all the members of the typhoid-coli group but not cocci; in the case of the artificial cholera stools nearly always pure cultures of *V. cholerae* were obtained.

Dieudonné's blood agar was found to permit the growth of *B. alcaligenes* and other coliform organisms in many cases. The agar of Esch allowed the growth of all organisms except Shiga's dysentery strain so that it seems to have only the slightest selective capacity.

Pilon's agar was effective in suppressing all vibrios but *V. cholerae*. These laboratory experiments indicated that Kabeshima's medium satisfied the conditions better than any of the others and he was able to confirm this in an actual epidemic of cholera where he compared the same media and found similar results, with this difference, that Pilon's medium definitely delayed the growth of the colonies of genuine *V. cholerae* on the plates.

W. J. P.

LOGIE (W. J.). **On the Inhibition of the Cholera-Red Reaction by Certain Nitrite-Destroying Organisms and on the Mutual Inhibition of *B. dysenteriae* (Flexner) and *V. cholerae* when grown together.**—*Jl. of Hygiene*. 1913. July. Vol. 13. No. 2. pp. 162-167.

Logie shows that if *V. cholerae* and *B. coli* be grown together and the culture be tested for the cholera-red reaction after twenty-four hours' growth, it may be absent. This he found due to the fact that the nitrite produced by the *V. cholerae* was destroyed by *B. coli*. If a little nitrite were added to the mixed culture the reaction was obtained. By testing for the presence of nitrite Logie was able to demonstrate its presence in the symbiotic cultures after a few hours' growth, but after 15 to 24 hours the whole of the nitrite is destroyed. *B. enteriditis* of GAERTNER has the same effect when grown in symbiosis with the *V. cholerae*. The *B. typhosus*, *B. paratyphosus* A and also B, the *B. dysenteriae* (Flexner), though they destroy nitrites, fail to do so to any extent when grown in symbiosis with the *V. cholerae*, hence they do not abolish the cholera-red reaction. Logie has found this effect to be due to the mutual inhibition of the growth of the two organisms.

W. J. P.

PETRONI (Vittorio). **L'Azione di due Microbi dell' Aria sulle Proprietà Biologiche del Vibrione Colerigeno.** [The Action of two Air-Organisms on the Biological Properties of the *Vibrio cholerae*.] — *Giorn. R. Soc. Italiana d'Igiene*. 1913. July 31. Vol. 35. No. 7. pp. 289-299.

The author draws attention to the modifications caused by sarcina in the agglutination, virulence and pigment producing power of the specific cholera germ. Two organisms obtained from air are fully described by him in the paper, which have also marked effect on the *Vibrio cholerae* when grown in symbiosis with it.

He found that, if plates on which the vibrios were growing were exposed to the air, in the neighbourhood of the colonies of the air organisms the colonies of the cholera strain assumed a brown colour tending to violet. Growth at air temperature is better for the pigment production than at 37° C. Filtrates of these air organisms have not this chromogenic power, and the actual strains tend to lose it in culture. The pigment is not destroyed by strong H<sub>2</sub>SO<sub>4</sub> or NaOH.

Two inagglutinable strains of *V. cholerae* were grown in symbiosis with these air organisms for five passages of about one week each, after which one was clumped by a 1:500 dilution of a serum of high titre; the other strain did not acquire the agglutinating power at all. Some evidence is given that these organisms cultivated with the cholera germ increase its virulence.

W. J. P.

**HOROWITZ (L.). Zur Frage über Cholera-Toxine und Antitoxine.**  
[On the Question of Cholera-Toxin and Antitoxin.]—  
*Zeitschr. f. Immunitätsforsch. u. exper. Therapie.* 1913.  
Aug. 5. Vol. 19. No. 1. pp. 44-65.

The paper is of a very diffuse character. After an introductory survey of the literature of this question the author proceeds to her own results.

The media used for the cultivation of the *V. cholerae* were broth, serum-broth, glucose-broth and the contents of the small intestine of a dog obtained from a suitable fistula: the dog was fed on casein before the samples were taken. The filtrates of these various broths were tested on guinea-pigs. The glucose-broth filtrate was found to be the most toxic and Horowitz was able to convince herself that the toxicity of the filtrate was not proportional to the vitality of the culture, but rather to the number of dead bacteria present in it. The glucose-broths were sterile on the third day. Marked acidity developed in the cultures and the acid produced is believed by Horowitz to be a much more delicate extracting agent than those commonly employed. In this way extracts were obtained fatal to guinea-pigs in  $\frac{1}{2}$  cc. doses. Evidence is brought forward to show that the toxicity of these solutions is due to endotoxin. It is heat-stable, withstanding boiling for 15 to 20 minutes.

Two animals immunised with the above filtrate showed that serum could be produced which could remove the toxicity of the broth filtrates; the serum so obtained however had very low agglutinating and bacteriolytic powers.

Whole glucose broth cultures of 3 or 4 days age produced sera which had high agglutinating and bacteriolytic titres and had also the power in fair degree of destroying the toxicity of filtrates. Sera on the other hand made by immunizing the rabbits with heated agar cultures had high agglutinating and bacteriolytic titres but did not destroy to any great extent the toxicity of filtrates. Horowitz believes the toxin-destroying body of these serums is not the same as the bacteriolysin but is related to it as a peptolytic is to a proteolytic ferment.

In experimental infections of animals a serum made by the injection of heated cholera vibrios is not so efficacious as a serum made by the injection of a three or four day old broth culture.

The destruction of toxin by any of the sera mentioned is a complement action. To obtain the measure of this power possessed by any serum, one must use the filtrate as toxin and not vibrios, since their lysis uses up the complement, and so hides the

toxinolytic effects of which the serum is capable in the presence of complement.

The effect of *Sarcina lutea* in raising the virulence of the *V. cholerae*, first pointed out by METCHNIKOFF, has been confirmed by Horowitz.

W. J. P.

POTTEVIN (Henri). *Toxine et Antitoxine Cholériques.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 409-413.

The author reviews the main results obtained by different workers up to the present with cholera toxins. He believes that his results reconcile to some extent the conflicting opinions.

He used for his work three vibrios: one vibrio A, isolated in Italy, and a second one identical with A, No. 70 of the Berlin collection; these two produced a toxin which is rapidly fatal to young rabbits and pigeons when injected intravenously and is strongly haemolytic. Both toxin and haemolysin are thermolabile. One hour at 50° C. destroys the haemolysin totally and the toxin in great part. The toxic effect that is left is not removed even by prolonged heating at 100° C. The heat-stable fraction has only 1/4 to 1/8 of the toxic power of the original and does not affect pigeons at all. The third *Vibrio cholerae*, isolated in Constantinople in 1913, gave a toxin similar to that described by ROUX, METCHNIKOFF and SALIMBENI. Intravenously in rabbits it caused death but never suddenly irrespective of dose. It is inactive in pigeons and is relatively thermostable. Immunization carried out in animals produced an antitoxin to the thermolabile poison, but no antibody to the thermostable fraction could be shown to exist in the serums produced by immunizing with either type of organism.

W. J. P.

POTTEVIN (H.) & VIOLETTE (H.). *Sur les Vibrions et leur Toxines.* [On the Vibrios and their Toxins.]—*Compt. Rend. Acad. Sciences.* 1913. June 30. Vol. 156. No. 26. pp. 2029-2031.

The authors describe a *Vibrio* isolated from the water of the Seine at Saint Cloud this year which gave all the characters of Koch's comma bacillus including positive agglutination and Pfeiffer's test. The organism produced a toxin and haemolysin.

The authors were able to absorb the haemolysin by red cells and to leave the toxin but slightly weakened, so that they believe the two to be distinct. They further show that by subdural administration sudden death is induced by a thermolabile poison without any haemolysis occurring.

W. J. P.

CRASTER (C. V.). *The Properties and Agglutinations of some Non-pathogenic Vibrios.*—*Jl. of Infectious Diseases.* 1913. May. Vol. 12. No. 3. pp. 472-480. With 4 text-figs.

Craster gives an account of over 100 non-cholera vibrios. They were obtained in the course of routine examinations at the Quarantine Station, New York. Compared with *V. cholerae* morphologically they may be similar or may show long forms with blunt

ends or again short forms with pointed ends. They are all mono-flagellates and highly motile. In alkaline broth and peptone water they form clouds and pellicles, as *V. cholerae*. They liquefy gelatine as a rule more quickly than *V. cholerae*. They are haemolytic, and Craster suggests this differentiates them from *V. cholerae*, but that view is not very general now. They ferment sugars similarly to *V. cholerae*. In the case of no strain was a typical cholera-red reaction given. They form colonies on plates which are more globose and granular than those of cholera, and are inclined to turn up when touched with the platinum loop while cholera colonies are inclined to run.

They have not such a faecal odour as genuine cholera cultures. Three of these vibrios were pigment-formers and one was a gas-former. None were pathogenic to guinea-pigs intraperitoneally in doses up to 1 agar slope.

None of these strains agglutinated with a genuine cholera serum to  $\frac{1}{10}$  dilution. Twenty of them were used to make special sera and the interagglutinations of these twenty members of the group were worked out. All the sera had very low titres and a positive reaction at  $\frac{1}{10}$  was taken as standard.

W. J. P.

PUNTONI (Vittorio). **I Vibrioni "Inagglutinabili." Loro Rapporti con il Vibrione Colerigeno e loro Importanza nella Eziologia e Profilassi del Colera.** [The Inagglutinable Vibrios.]—*Polislinico. Sez. med.* 1913. Sept. Vol. 20. No. 9. pp. 385-409.

The history of cholera diagnosis from 1883 to the present day is discussed and the variability of the cultural and biological tests insisted upon. An account is given of attempts with different genuine strains to produce inagglutinable vibrios from them by growth in water. Puntoni was able in this way to get several positive results. He removed also, by growing in earth and water mixtures, the agglutinating power of several vibrios. Why the methods were not uniformly successful he has not ascertained.

He finds these degenerated vibrios produce, in the animal body, agglutinins, which do not react with undoubted strains. Towards the end of an epidemic the vibrio may become modified so that it does not present itself with the classical characters. The transformation of cholera vibrios into cholera-like vibrios must now be accepted, and consequently the cholera-like vibrios isolated from patients and the healthy must be looked upon as simply a saprophytic variety of the real cholera organism.

From the standpoint of prophylaxis the inagglutinable vibrios require to be considered seriously.

W. J. P.

POTTEVIN (H.) & VIOLE (H.). **Transmission du Choléra aux Singes par la Voie Gastro-intestinale.**—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 482-484.

Starting from the clinical fact that purgatives predispose to cholera the authors have used this method to infect monkeys (*Cynomolgus*).

They first administer sulphate of soda; after eight hours, when purgation has commenced, a dose of sodium bicarbonate is given, then after twenty minutes the cholera culture. Death follows in 12-48 hours. The controls with the same treatment minus the cholera culture remain quite well. In order to get positive results a toxogenic cholera strain must be employed.

W. J. P.

POTTEVIN (H.) & VIOLLE (H.). *Choléra expérimental des Singes inférieurs.*—*Compt. Rend. Acad. Sciences.* 1913. Aug. 4. Vol. 157. No. 5. pp. 343-345.

The authors repeat in this paper the statement that they have produced experimental cholera in monkeys (*cynomolgus* and *rhesus*). The animals die in 12 to 48 hours. They find, however, that if cultures (killed by ether) of the cholera organism be used they get practically the same effect. By diminishing the dose death does not occur until two to four days after the administration of the organisms. They do not say, however, whether this is a genuine infection or simply an intoxication. A little of the intestinal contents of a monkey which had died from the administration of the culture was fatal to other monkeys. The disease produced is similar in all essential points to human cholera.

An antitoxic and bacteriolytic serum prepared by means of the vibrio used prevents the death of the animals if given a few days before they receive the cultures. The first attack of the disease confers immunity against subsequent attacks.

W. J. P.

PANAYOTATOU (Angélique). *Survie du Vibriion Cholérique dans l'Eau du Nil.*—*Rev. d'Hyg. et de Police Sanitaire.* 1913. July. Vol. 35. No. 7. pp. 779-787.

In spite of carriers the essential agent in the spread of cholera is infected water. Panayotatou surveys the literature up to the present dealing with the bactericidal effect of different waters and shows how strikingly divergent the results obtained have been; HAFKINE finding the Indian waters destroy the vibrios in a few hours and other workers discovering them alive after as many as 392 days.

The author believes water which is not contaminated by the specific vibrio may yet play a part in the causation of cholera, in so far as it may contain other organisms which favour the growth and exalt the virulence of the vibrios occurring in healthy carriers. She favours the study of the effect of local waters on the cholera germ under diverse conditions.

The Nile water at laboratory temperature destroys added comma bacilli in from 24 hours to 13 days. The bactericidal effect varies with the season and the precise place where the water is drawn.

The sterilization of the water destroys its bactericidal effect. The cause of the bactericidal effect is partly due to toxins secreted by the bacteria but chiefly to the actual symbiosis with certain microorganisms. Panayotatou isolated nineteen varieties of bacteria from Nile water, and was able to show that four of these were highly antagonistic to the comma bacillus while many were indifferent.

W. J. P.

#### PROTECTIVE INOCULATION.

HAFFKINE (W. M.). **Protective Inoculation against Cholera.**—98 pp. 4to. Illustrated. 1913. Calcutta: Thacker, Spink & Co. London: W. Thacker & Co. [Rs. 3, or 4s. 6d. net.]

This monograph should be read by all engaged in cholera vaccination in particular and vaccine work in general. It starts with an account of the variability of the *V. cholerae* and the bearing this has on cholera vaccination. The inter-relationship of virulence and immunizing power is discussed and the outstanding data in the literature of this subject are reviewed. The history of cholera vaccination in Spain and the reasons of the doubtful character of the results of the same are discussed, though it must be admitted not quite convincingly. The methods used to raise the virulence of the *V. cholerae* are described and, in considerable detail, as well as Haffkine's own method together with certain general principles to be followed in endeavouring to attain this end. The essential object of the research is then revealed. It was to ascertain whether by the use of Haffkine's vaccines an undoubted protection to cholera could be produced. The vaccines are living vaccines, and No. II. is of high and constant virulence. A complete account of the immunization carried out by Haffkine of certain sample populations in India against control populations is then given, and it appears to the reviewer that we have here a model of how such work should be arranged and performed.

The control populations lived in the same houses, barracks, gaols, ships, etc., as the inoculated, and were as far as possible similar. The results of the inoculations in Calcutta were as follows:—Three entirely different post-inoculation periods had to be recognised:

(1) The first four days after vaccination, during which the mortality as between the inoculated and the controls is but slightly different.

(2) The 5th to the 416th day after inoculation where the mortality of the uninoculated was 11·32 per cent. of the exposed, against 72 per cent. of the inoculated and equally exposed.

(3) From the 417th day onward the inoculated were found to be in a post-immune period during which they possessed no protection against death. A very striking result of the experiment was to show that the immunization protected against the incidence of the disease but did not definitely diminish the case mortality. The number involved in the above and similar experiments in



different parts of India, and the care taken to check all the data, give us great confidence in the results of the work.

The monograph concludes with a discussion of the question of using vaccine II. in a devitalized condition.

W. J. P.

LEOPOLD (L.). *Het Choleravaccin te Stagen*. [Cholera Vaccination in Stagen.]—*Geneeskundig Tijdschr. v. Nederl.-Indië*. 1913. Vol. 53. No. 3. pp. 475-478.

The first part of this short note is taken up with criticism of previous work on the question. The results of 1,297 vaccinations are given. Fifteen of the vaccinated took the disease *i.e.* 1.16 per cent., whereas only 1.12 per cent. of the 1,071 controls took it. The case mortality of the vaccinated patients was however only  $33\frac{1}{3}$  per cent. against 50 per cent. in the unvaccinated.

W. J. P.

## DYSENTERY.

## AMOEBIASIS AND AMOEBIC DYSENTERY.

CRAIG (Charles F.). **The Relation of Parasitic Amoebae to Disease.**  
—*War Dept. Office of the Surgeon-General. Bulletin No. 2.*  
1913. Jan. pp. 95-113.

In this paper the author gives an excellent and up to date account of the parasitic amoebae. He commences with mention of the earlier works which led to the adoption of the classification of the amoebae now in use, he then discusses at length the relation of the entamoebae to disease, giving in detail many experiments carried out by himself and other investigators on animals.

The paper is concluded by a discussion on the relation of cultural amoebae to disease.

The author's conclusions are as follows:—

1. *Entamoeba coli* is a harmless commensal in the human intestine.

2. *Entamoeba histolytica* and *Entamoeba tetragena* are pathogenic species capable of producing in men the disease known as amoebic dysentery. *E. tetragena* is now believed to be identical with *E. histolytica*.

3. *Entamoeba coli*, *Entamoeba histolytica* and *Entamoeba tetragena* are strictly parasitic species and have not been cultivated.

4. There is not sufficient evidence at present to prove that any of the amoebae that have been cultivated are pathogenic to man. All cultivated species belong to the genus *Amoebae* and differ greatly in morphology and life cycle from the parasitic amoebae, which belong to the genus *Entamoebae*.

An excellent list of references is given.

S. R. Douglas

CRAIG (Charles F.). **The Identity of *Entameba histolytica* and *Entameba tetragena*, with Observations on the Morphology and Life-Cycle of *Entameba histolytica*.** — *Jl. of Infectious Diseases*. 1913. July. Vol. 13. No. 1. pp. 30-52. With 2 plates.

The first part of the paper is occupied with a series of extracts from various authors to show their opinions on the plurality of species of pathogenic entamoebae. Craig then summarises his results of over 1,000 cases of amoebic dysentery, classifying the types of amoeba and virulence of cases into five groups. From these he concludes that:—

1. Entamoebae with the histolytica type of nucleus are most common in cases with severe symptoms and are characteristic of entamoebae undergoing rapid, simple division. They are not degenerative.

2. Entamoebae with typical tetragena nuclei are most frequent in cases with slight symptoms and are characteristic of *E. histolytica* when undergoing simple division preparatory to forming the small generation that produces cysts.

3. Entamoebae reduced in size and with generally the tetragena type of nucleus occur in chronic cases. They are apparently characteristic of *E. histolytica* preparatory to cyst formation.

4. Cysts with four nuclei occur most frequently in cases that have apparently recovered.

5. Entamoebae showing appearances interpreted as budding or spore formation occur most frequently in cases with subacute symptoms. They are believed to be degenerative in nature.

The life cycle of *Entamoeba histolytica* is then considered in some detail. It is divided into three main stages, each of which is discussed. They are:—"A vegetative stage of development in which multiplication occurs rapidly by simple division: a pre-cystic stage in which multiplication occurs by simple division and the entamoebae become markedly reduced in size; and a cystic stage during which cysts containing four nuclei are developed."

The author concludes by discussing nomenclature, stating that the identity of *E. histolytica* and *E. tetragena* having been established, the latter name no longer has specific rank.

A. Porter.

MATHIS (C.). Recherche des Kystes d'Amibes dans les Selles de l'Homme.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*. 1913. July. Vol. 4. No. 7. pp. 334-350. With 4 plates.

The present paper aims at giving indications that may be of service in dysenteric cases at periods when encystment of the amoebae has occurred.

1. *Examination of fresh stools*. Cysts of dysenteric amoebae are distinguished from *Entamoeba coli* from the colon, by their size, the former cysts being  $12.5\mu$  to  $14.5\mu$  in diameter, the latter  $16\mu$  to  $19.5\mu$ . Four nuclei are present in the mature dysenteric cyst, 8 in that of *E. coli*.

2. *Examination of fixed and stained preparations*. Double infections are common. Cysts with four nuclei may be mature *E. histolytica*, or immature *E. coli*. Distinguish between them by size. Dysenteric cysts also often contain chromidial blocks.

3. *Elimination of cysts in faeces*. Cysts appear at the decline of a dysenteric attack, especially when the faeces regain their normal consistency. Cysts can persist for years in old dysenteric cases.

4. *Differential diagnosis of Entamoebic cysts from those of other Protozoa and Blastocystis hominis*.

(a) Cysts of *Amoeba limax* type. These are rare. They have a double contour, the outer polygonal, the inner rounded.

(b) Cysts of *Lamblia intestinalis* are ovoid,  $10\mu$  to  $15\mu$  by  $8\mu$  to  $9\mu$ . One or more clearly curved lines occur along the long diameter of cyst. Giemsa-stained smears are useful for diagnosis, as they stain blue with 3 or 4 violet-red nuclear masses at the anterior summit.

(c) Cysts of *Prowazekia weinbergi* are common. Cysts are  $5\mu$ - $7\mu$  diameter, and round. Their envelope is very thin. They are extremely refringent.

(d) *Blastocystis hominis*. The fungus shows as spherical bodies chiefly,  $10\mu$  to  $15\mu$  diameter. They consist of a central, absolutely homogeneous, rounded mass, within a clearer, less refringent layer in which a variable number of nuclei are present. Mathis's observations are restricted entirely to cases observed in Indochina. Ha

points out the importance of the search for cysts for prophylactic measures, in retrospective diagnosis of dysentery, and in judging the value of various medicaments used. The plates illustrate *E. coli*, *E. histolytica*, and *Lambliia intestinalis*.

A. P.

**DARLING (S. T.). The Rectal Inoculation of Kittens as an Aid in determining the Identity of Pathogenic Entamoebae.**—*Southern Med. J.* 1913. Aug. Vol. 6. No. 8. pp. 509-511.

Darling's present paper covers much the same ground as those previously reviewed in this *Bulletin* (Vol. 1, pp. 180, 462, 720, and Vol. 2, p. 165). The rectal inoculation of fresh dysenteric material into kittens does not produce degenerative changes in the entamoebae. The author describes fully the morphology of the dysenteric trophozoite which "represents something analogous to adolescence," the precyst trophozoite, corresponding to *E. minuta* of ELMASSIAN, and the cyst representing either maturity or senility. An adolescent strain can be carried through as many as six transfers; a senile form cannot be maintained for more than one or two. All intermediate types from typical "histolytica" to typical "tetragena" forms can be obtained by successive passages through kittens. Pathological specimens of amoebae show budding, extrusion of chromidia and differences in staining properties. The writer believes that cases thought to be "histolytica" dysentery will resolve themselves into infections by *E. tetragena*.

A. P.

**JAMES (W. M.). The Clinical Identification of Entamoebae.**—*Proc. Canal Zone Med. Assoc.* 1912. Vol. 4. Pt. 2. [12 pp.]

James indicates his plan for determining, for diagnostic purposes only, the identification of *Entamoeba coli*, *E. histolytica* and *E. tetragena* in fresh preparations in Panama. He relies chiefly on the character of the nucleus, ectoplasm, endoplasm and rate of movement in diagnosis.

- Nucleus.** *E. coli*.—Distinct, with bright granules, always circular.  
*E. histolytica*.—Not so prominent, irregular ring or ellipse.  
*E. tetragena*.—Centriole present.
- Endoplasm.** *E. coli*.—Well marked, always grey.  
*E. histolytica* and *E. tetragena*.—Greenish if stools contain erythrocytes, grey otherwise.
- Ectoplasm.** *E. coli*.—Lobose pseudopodia, ectoplasm only distinct for a short time.  
*E. histolytica* and *E. tetragena*.—Long rapid, finger-like pseudopodia, ectoplasm distinct relatively long time.
- Movement.** *E. coli*.—Crosses field of microscope relatively slowly compared with *E. histolytica* and *E. tetragena*.

The differences in the cysts are mentioned only. Cysts of *E. coli* are said to be  $12\mu$  to  $18\mu$  diameter, those of *E. tetragena*  $10\mu$  to  $14\mu$ . Neither spores nor buds of *E. histolytica* were seen by the writer in cases in Panama.

[Apparently Dr. James does not believe in the identity of *E. histolytica* and *E. tetragena* from his diagnostic characters.]

A. P.

GIFFIN (H. Z.). **Clinical Notes on Patients from the Middle North-west infected with Entamebas.**—*Jl. Amer. Med. Assoc.* 1913. Aug. 30. Vol. 61. No. 9. pp. 675-677.

During the last two and a half years the author has examined the stools of 1,700 patients and has discovered entamoeba in 227 cases. Of these 148 were *E. coli* and 79 either *E. tetragena* or *E. histolytica*.

Of the 79 cases harbouring *E. tetragena* or *E. histolytica* 41 were patients residing in the middle north-west states.

The symptoms complained of by these patients were usually not very severe, such as attacks of diarrhoea often with the passage of blood, abdominal pain, indefinite gastric symptoms, etc.

Only one case of liver was observed.

Of the 148 cases in which *E. coli* was found, 106 were in patients residing in middle north-west, and about one half of these complained of diarrhoea; the author believes, if further examinations had been made, *E. tetragena* or *E. histolytica* would have been discovered in such cases.

Treatment employed was usually ipecacuanha, and the author evidently has a belief that enemata of kerosene oil are efficacious.

S. R. D.

BAERMANN (G.) & HEINEMANN (H.). **Die Behandlung der Amöben-Dysenterie mit Emetin.** [The Treatment of Amoebic Dysentery by Emetine.]—*München Med. Wochenschr.* 1913. May 27. Vol. 60. No. 21. pp. 1132-1135; and No. 22. June 3. pp. 1210-1213.

The authors give a summary of the researches of WHERRY, VEDDER and ROGERS, and then go on to describe their own clinical experiences with the method. The supply of emetine available being but small, only 22 cases could be so treated, of whom seven died from intercurrent complications during observation. The post mortem examinations in these cases enabled the authors to note the remarkable improvement in the local lesions brought about by the drug. The dose of hydrochloride\* of emetine for hypodermic injection seems to lie between 150 and 200 milligrammes, but for intravenous injection it may vary from 60 to 200

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\* The terms hydrochlorate and hydrochloride of emetine are synonymous. Salts formed by the combination of an alkaloid with hydrochloric acid were in this country formerly called hydrochlorates, but of late years they have been commonly known as hydrochlorides. In France however the term hydrochlorate is still in use for such salts.—S. R. D.

milligrammes, according to the indications. Larger doses are apt to induce unpleasant collapse, so that 250 milligrammes should be considered the maximum dose for a body-weight of 60 kilos.

Though the general results were most favourable as regards the healing of ulcers and the amelioration of the clinical symptoms, the authors could not help noticing a tendency to relapse in a certain number of cases, indicated by the re-appearance of amoeba in the stools after an interval of ten days or so. This should necessitate the repetition of the treatment and therefore patients should not be discharged from hospital too promptly, but in spite of this drawback, the treatment seems to surpass in effectiveness all means hitherto tried for the relief of amoebic dysentery.

S. R. D.

ROUX (G.) & TRIBONBEAU (L.). *Action de l'Éméline dans Quelques Formes spéciales d'Amibisme et, par Analogie avec une d'elles, dans la Syphilis.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 424-427.

The account of four cases. The first was one of dysentery in which the stools were found to contain amoebae and also large numbers of spirilla, treatment with emetine caused the disappearance of both the spirilla and amoebae.

The second case was one of dysentery in which amoebae and *Trichomona vaginalis* were said to be demonstrated in the stools, treatment with emetine caused the disappearance of the amoeba but had no effect on the *Trichomona* and the diarrhoea continued.

The third case was one of multiple abscesses of the liver which rapidly improved under the emetine treatment.

The fourth case is one of primary syphilis in which the authors having noted the disappearance of spirilla from the stools during treatment with emetine, determined to try the effect of this drug on the *Spirochaeta pallida*. The result was negative.

S. R. D.

GAIDE (L.) & MOUZELS (P.). *Note sur le Traitement de la Dysenterie Amibienne par l'Éméline.*—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 491-494.

The report of four cases of amoebic dysentery in which, although the symptoms and general condition of the patient improved rapidly under treatment with emetine, cysts and in one case active amoebae could always be demonstrated in the stools.

S. R. D.

JOB (E.) & LÉVY (L.). *Un Cas de Dysenterie Amibienne chronique traitée par l'Éméline.*—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris.* 1913. May 15. (3 sér.) Vol. 29. No. 16. pp. 988-993.

A report of a case of severe chronic amoebic dysentery treated with subcutaneous injections of emetine hydrochloride. Rapid recovery.

S. R. D.

**DESTÉFANO (José).** **Dos Casos de Disenteria tratados con el Método de Rogers.** [Two Cases of Dysentery treated by the Method of Rogers.]—*Semana Medica*. 1913. May 22. Vol. 20. No. 21. pp. 1189-1192.

An account of the treatment at Buenos Aires of two cases of amoebic dysentery, one acute and the other chronic, by injections of emetine. 0.04 gm. of the hydrochloride was given night and morning, with excellent results in each case.

S. R. D.

**MAXWELL (J. Preston).** **The Use of Emetine Salts in the Treatment of Amoebic Dysentery.**—*China Med. Jl.* 1913. March. Vol. 27. No. 2. pp. 116-119.

The report of ten cases of amoebic dysentery treated by the subcutaneous injections of emetine hydrochloride. The doses given were one to five centigrams daily and the result in each was very satisfactory.

S. R. D.

**HUTCHESON (Allen C.).** **Results in Thirteen Cases of Dysentery treated with Emetine.**—*China Med. Jl.* 1913. July. Vol. 27. No. 4. pp. 243-245.

The cases reported were mostly uncomplicated amoebic dysentery, all of which rapidly improved; besides these, two were infected with amoeba and *Schistosoma japonicum*, one of which improved markedly under the treatment. Three other cases infected with *Schistosoma* only were treated, two of which showed marked improvement of symptoms.

S. R. D.

**LA CAVA (F.).** **La Chemioterapia della Dissenteria da Amebe. Due Casi Curati col Cloridrato di Emetina secondo il Metodo di L. Rogers.** [The Drug Treatment of Amoebic Dysentery. Two Cases cured with Emetine Hydrochloride after Rogers' Method.]—*Malaria e Malatt. d. Paesi Caldi*. 1913. Apr.-May. Vol. 4. No. 3. pp. 189-197.

Observations on the subject, and an account of two cases satisfactorily cured with doses ranging from one quarter to one decigramme.

S. R. D.

**Low (George C.).** **The Administration of Emetine by the Mouth in Amoebic Dysentery.** [Memoranda.]—*Brit. Med. Jl.* 1913. June 28. pp. 1369-1370.

The account of a case of chronic amoebic dysentery treated with keratin-coated tabloids of emetine hydrochloride, each of which contained half a grain of the emetine salt. The dose administered was one tabloid every evening.

The improvement was very rapid and no nausea or other unpleasant symptoms were noticed.

S. R. D.

THOMPSON (J. H. C.). **The Treatment of Dysentery by Injections of Emetine Hydrochloride.** — *Dublin Jl. of Med. Sciences.* 1913. Aug. 3. No. 500. pp. 102-109.

A thesis in which the author gives an account of the work which led to the use of emetine as a cure of amoebic dysentery, and the details of seven cases successfully treated by the subcutaneous injections of this drug.

S. R. D.

LYON (Gaston). **Traitement par l'Émetine de la Dysentérie amibienne.**—*Revista Med. de S. Paulo.* 1912. Oct. 31. Vol. 15. No. 20. pp. 406-408.

The author gives a resumé of some of the literature on the treatment of dysentery by the subcutaneous injection of emetine salts. No original observations are quoted.

S. R. D.

CHAUFFARD (A.). **Les Mauvais Effets de l'Émetine en Lavement dans la Dysenterie Amibienne.**—*Bulls. et Mém. Soc. Méd. des Hôpit. de Paris.* 1913. July 3. (3 ser.) Vol. 29. No. 23. pp. 1235-1237.

The report of a case of chronic dysentery which rapidly improved under treatment with subcutaneous injections of emetine hydrochloride, but the stools remained semi-solid and to remedy this condition an enema composed of ten centigrams of emetine hydrochloride dissolved in a litre of water was administered. This was immediately followed by a return of the dysenteric symptoms, fourteen motions containing blood and mucous being passed in the next 24 hours, the recrudescence was however of very short duration and the patient rapidly recovered.

S. R. D.

MAURIN (M.). **Dysenterie Amibienne Traitée et Guérie par la Décoction d'Ipéca en Lavements.**—*Bulls. et Mém. Soc. Méd. des Hôpit. de Paris.* 1913. July 31. (3 ser.) Vol. 29. No. 27. pp. 282-285.

The author reports a case of dysentery which rapidly recovered when treated with enemata of a decoction made of four grams of ipecacuanha in 200 cc. of water, and in addition, six grams of bismuth were administered daily by the mouth.

S. R. D.

WADHAM (S. H.) & HILL (E. C.). **Three Cases of Amoebic Dysentery treated with Salvarsan.**—*Jl. Amer. Med. Assoc.* 1913. Aug. 9. Vol. 61. No. 6. pp. 385-386.

The authors give details of three cases in which all the symptoms of dysentery and the amoebae in the stools disappeared after administration of neo-salvarsan. In two cases the drug was given intravenously and in one intramuscularly.

S. R. D.



**DENIER & HUET.** *La Dysentérie à Saïgon.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 413-415.

The authors report the results of their examination of the stools in 23 cases of dysentery occurring in Saïgon between May and October 1912.

In seven cases dysentery bacilli were isolated, three belonging to the Shiga type, two to the Flexner type and from two cases, both young children, bacilli resembling the type Y and type Saïgon were obtained.

In ten of the remaining sixteen cases amoebae were demonstrated.

S. R. D.

**WHITMORE (E. R.).** *Dysentery in the Tropics.*—*New York Med. Jl.* 1913. Aug. 9. Vol. 98. No. 6. (Whole No. 1810.) pp. 257-260. With 3 figs.

A general paper on dysentery both bacillary and amoebic which contains no new information.

S. R. D.

**LUKIS (Charles Pardey).** *An Address on the Diagnosis and Treatment of Dysentery.* (Delivered before the South Midland Branch of the British Medical Association.)—*Brit. Med. Jl.* 1913. June 28. pp. 1357-1359.

The author gives in his address an excellent account of both amoebic and bacillary dysentery. The points discussed included the differential diagnosis between bacillary and amoebic dysentery, the diagnosis of liver abscess and the treatment of the manifestations of both types of the disease.

S. R. D.

#### LIVER ABSCESS.

**PERVÈS & OUDARD.** *Une Série de Vingt Cas Personnels d'Abscess du Foie des Pays Chauds.*—*Arch. de Méd. et Pharm. Navales.* 1913. Apr. Vol. 99. No. 4. pp. 241-270; and May. No. 5. pp. 321-353.

In this paper the authors give the complete clinical descriptions of twenty cases of liver abscess, discussing also the operations performed for the evacuation of the pus, any complications that occurred, morbid anatomy, etc. Amongst other conclusions the authors believe that—

1. Liver abscess occurs frequently in individuals returning from the tropics, and when the following symptoms are present a liver abscess should be suspected:—

(a) Enlargement of the liver with a sense of weight in the liver region.

(b) A point in that region which is distinctly painful to pressure.

(c) The previous history of dysentery.

2. The cases seen in France are generally of the chronic type.

3. The absence of polymorphonuclear leucocytosis does not contra-indicate the presence of an abscess.

4. The presence of glycogen in pus free from blood indicates that the abscess is situated in connection with the liver.

5. Exploration by aspiration is a sound method and gives satisfactory results.

6. Ether should be used as an anaesthetic in preference to chloroform in such cases.

7. A frequent pulse-rate (100-120) after the operation is not necessarily a bad sign.

8. The escape of bile occurring from the drainage tube is not as a rule a serious complication.

S. R. D.

CARDARELLI (A.). **Come si Diagnostica l'Ascesso Epatico.** [The Diagnosis of Hepatic Abscess.]—*Riforma Medica*. 1913. June 21. Vol. 29. No. 25. pp. 791-796.

A clinical lecture upon a single case, without any particular novelty.

S. R. D.

CHAUFFARD (A.). **Abcès Dysentérique du Foie, avec Vomiques successives. Traitement par la Ponction évacuatrice et l'Éméline.**—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris* 1913. May 22. (3 sér.) Vol. 29. No. 17. pp. 1017-1024.

The report of a case of liver abscess which had burst into the lung about 12 months before being admitted into hospital under the author's care. The treatment employed was aspiration and administration of emetine hydrochloride subcutaneously: the patient improved rapidly, this improvement the author believes to be due more to the aspiration than to the emetine as no amoebae were discovered in the pus.

S. R. D.

VALENCE (M.). **Abcès du Foie traité selon la Méthode de Rogers. Cures d'Éméline contre l'Amibiase.**—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*. 1913. July 31. Vol. 29. (3 sér.) No. 27. pp. 276-282.

The report of a case of chronic liver abscess treated by aspiration of the pus, injection into the abscess cavity of a solution of emetine hydrochloride together with the subcutaneous injection of emetine. The patient rapidly improved.

S. R. D.

COUTEAUD. **La Chirurgie l'Éméline et l'Epeca dans le Traitement des Abcès du Foie.**—*Bull. et Mém. Soc. de Chirurg. de Paris*. 1913. June 17. Vol. 39. No. 22. pp. 941-949.

This paper contains a complete account of two cases of liver abscess, one of which was treated by aspiration and the subcutaneous injection of emetine, the other by incision and drainage together with the lavage of the abscess cavity with a decoction of ipecacuanha. Both cases recovered.

S. R. D.

MALLANNAH (S.). **Emetine and Liver Abscess.** [Correspondence.] — *Indian Med. Gaz.* 1913. Aug. Vol. 48. No. 8. pp. 331-332.

A report of a case which has already been noted in this *Bulletin*, Vol. 2, p. 170, as it was published in full in the *British Medical Journal*, 1913, June 7, pp. 1206-1207.

S. R. D.

GLUZET & BAUR (J.). **Hydropneumocyste hépatique au Cours d'un grand Absès du Foie, consécutif à une Dysenterie, examens Radioscopiques et Radiographiques.** — *Lyon Médical.* 1913. July 13. (45 année.) pp. 50-55. With 2 plates.

An account of a case of liver abscess, in which, after aspiration, the cavity of the abscess was found by examination with x rays to be filled with gas.

The authors discuss the origin of the gas and believe it to have been allowed to enter through the aspirating trocar during the introduction of a solution of emetine into the cavity.

S. R. D.

SAMRUC (E.). **Les Absès du Foie à l'Hôpital de Haiphong.** — *Arch. Générales de Chirurgie.* 1913. June 25. Vol. 7. No. 6. pp. 641-659.

This paper has already been noticed in this *Bulletin*, Vol. 2, pp. 167-169, as it has already appeared in the *Annales d'Hygiène et de Médecine Coloniales*.

S. R. D.

#### BACILLARY DYSENTERY.

EBELING (E.). **Beobachtungen über die Y-Buhr, gelegentlich einer Epidemie.** [Observations on Y-Dysentery, with an account of an Epidemic.] — *Zeitschr. f. Hyg. u. Infektionskr.* 1913. June. Vol. 74. No. 3. pp. 447-472. With 1 plate.

An account of an extensive epidemic of dysentery due to the Y-bacillus, which attacked the cavalry of the 10th German Army Corps during the summer manoeuvres of 1911, in Hanover. Altogether 119 men presented unmistakable symptoms of the disorder while 390 more could be classed as suspects. The mortality was nil. The source of the infection could not be definitely traced, but the manoeuvre ground had been occupied throughout the summer by successive bodies of cavalry; and it is suggested that flies breeding in manure heaps which had been allowed to accumulate in the lines during the hot weather, formed the means of transmission. The pressing calls of nature, amongst men on guard affected with the disease, cause such manure-heaps to be used as latrines, inevitably, especially at night. This is a point always worthy of consideration, when investigating the causes of such epidemics. The organism isolated from the stools of patients was in every case the Y- or Hiss-Russell bacillus. A full account is given of its cultural and other characteristics, for which reference should be made to the original paper. A very

extensive examination of the stools of all contacts was carried out, with the result of discovering no less than 147 carriers of the disease among healthy men. The paper concludes with a number of recommendations with regard to camp-hygiene, of the ordinary type. Special care should be taken to clear away manure-heaps at shorter periods than ten days, this being the time occupied by the development of the ordinary fly from the egg.

S. R. D.

LORENTZ (F. H.). **Zur Dysenterie der Irrenanstalten.** [On Asylum Dysentery.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. May 23. Vol. 69. No. 3. pp. 113-132.

The author gives an account of an outbreak of dysentery in the asylum at Düren, which was only brought to an end by a systematic search for chronic dysentery-carriers among the patients. Four such were discovered, and by their isolation and the surveillance of all contacts the outbreak was finally suppressed. The author insists upon the necessity for equipping all large asylums with proper laboratories under the charge of an expert, in which the diagnosis of such diseases by modern methods can be carried out without delay. It should be a rule to pass all new patients through a quarantine block before distributing them throughout the asylum, and there should also be isolation-blocks wherein convalescents can be kept until it can be ascertained that they are not carriers. The uncleanly habits of the insane greatly facilitate the propagation of such diseases as dysentery in asylums, a fact of which the authorities in Germany do not seem sufficiently mindful; the type of dysentery seen in asylums often takes on a larval form so that it escapes recognition until too late. The mortality is always fairly high in spite of this fact, as the disease seems to have a preference for old and debilitated patients. It is a good thing to investigate all cases of febrile diarrhoea occurring in asylums upon the assumption that they are true dysentery, and serum-tests will generally give reliable indications for this purpose, though they are not absolutely conclusive as to the particular strain of the bacillus, according to the author's findings.

S. R. D.

BUTLER (C. S.). **Some Carbohydrate Reactions of the Dysentery Bacillus.**—*Philippine J. of Science.* Section B., Trop. Med. 1913. Apr. Vol. 8. No. 2. pp. 123-131.

The author insists on the importance of using media containing pure carbohydrates for the differentiation of the various types of dysentery bacilli, and lays special stress on procuring pure samples of maltose, as he believes that the use of impure samples of this sugar has led frequently to erroneous conclusions.

The media which the author has found most satisfactory, are very simple and are prepared as follows: -- Witte's peptone 1 per cent., the carbohydrates (purest obtainable) 1 per cent., sodium chloride (chemically pure) 0.5 per cent. dissolved in distilled water. The media are then distributed in test tubes, 10 cc. in each, and sterilized at 20 pounds pressure for 20 minutes.

Then to each tube 2 cc. of a 5 per cent. litmus solution which has been separately autoclaved is added, and the tubes are reesterilized in a steam sterilizer for fifteen minutes. Using such media the author obtained very consistent results, and appears to agree with the classification of the dysentery bacilli given by LEHMANN and NEUMANN. The following table shows the carbohydrate reactions.

Carbohydrate.	Shiga-Kruse.	Flexner.	Strong.	Bacillus Y.
Mannite ...	Blue ...	Red ...	Red ...	Red
Maltose ...	Blue ...	Red ...	Blue ...	Blue.
Saccharose ...	Blue ...	Blue ...	Red ...	Blue.

S. R. D.

BAUGHER (Albert Howard) & GAY (Robert S.). **An Epidemic of Bacillary Dysentery in Institutional Children.**—*Trans. Chicago Pathological Soc.* 1913. Feb. 1. Vol. 9. No. 1. pp. 8-10.

An account of an outbreak of dysentery occurring in the Chicago Orphan Asylum; during the epidemic 37 children, 3 caretakers and 1 laundress were attacked.

The disease was seemingly imported into the institution by a child who had had an attack of dysentery six weeks before admission.

The bacteriological investigations carried out by the authors appear to have been very incomplete and the conclusions arrived at unwarranted seeing that they state that the organism isolated from the infected stools "probably falls in the Flexner group" after having found that it produced acid and gas in media containing the following carbohydrates:—saccharose, dextrose, maltose, lactose, and mannite.

S. R. D.

#### BALANTIDIC DYSENTERY.

VON PROWAZEK (S.). **Zur Kenntnis der Balantidiosis.** **Zusammenfassende Darstellung.** [Summary of Literature of Balantidiosis.]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Aug. Beiheft 6. pp. 5-24. [pp. 371-390.] With 2 coloured plates and 9 text-figs.

The author gives a review of the work done on *Balantidium coli*, both in men and pigs, showing the balantidium to be definitely the cause of balantidium-infusorian-dysentery or balantidium-colitis. The parasite has been found in many parts of Europe and Asia, also in Africa and America. The size, as shown by accounts of various authors, lies between 60 $\mu$  to 200 $\mu$  long and 50 $\mu$  to 70 $\mu$  broad. The structure of the organism is discussed in detail, the various accounts extant being compared and amplified. The mouth is not terminal. The arrangement of the fibrillae is discussed, and the structure of the macronucleus in surface and section.

*Balantidia* from human *faeces* live about 30 hours as free organisms. According to GLAESSNER, *balantidium* possesses no proteolytic but a definite diastatic ferment. The parasite produces haemolysis.

Multiplication is by division, one individual forming a new peristome, the other retaining that of the parent. In the submucosa of patients, nests of *balantidium* and free spores have been found. The attempts at cultivation are set forth in detail.

Clinically, *Balantidium coli* causes much alteration in the blood. The pathological anatomy is reviewed, the mucosa and submucosa of the large intestine being chiefly infected. Accounts of therapeutic treatment by various workers are given.

*Balantidium coli* in pigs is very common. Swineherds and pig slaughterers appear most often as patients for *balantidium* dysentery. Cysts of various sizes appear frequently in pig *faeces*. Only one successful culture, that in physiological salt solution, has been made, other media have given negative results.

[To those interested, this paper gives a full review of the very scattered literature relating to *balantidium*, and should be consulted in the original.]

A. P.

#### SPRUE.

WEGELE (C.). Ueber die diätetische Behandlung gewisser Formen chronischer Diarrhöen speziell von "Indian Sprue." [The Dietetic Treatment of Certain Forms of Chronic Diarrhoea, and especially of Indian Sprue.]—*Medic. Klinik*. 1913. June. Vol. 9. No. 22. pp. 866-868.

A recommendation of the diet proposed by VAN DEN SCHEER (*Tijdschrift voor Geneeskunde*, 1905, No. 10) for sprue, consisting of fresh strawberries, with or without milk and other things. The author had the opportunity of treating in this way two cases of sprue returned from the tropics, one from Celebes and the other from Jamaica. To both fresh strawberries were given, first only the juice passed through a sieve, and mixed with lukewarm milk in the proportion of one part of juice to two of milk, and afterwards the fruit itself up to three pounds daily. From two to four eggs per diem were also allowed, with sponge-cake and bread and milk, as the condition of the alimentary canal improved. A good result with subsidence of all symptoms was obtained in both cases, which seemed to be permanent from subsequent communications received from the patients. The fresh fruit alone produced the desired effect.

S. R. D.

ASHFORD (Bailey K.). Notes on Sprue in Porto Rico and the Results of Treatment by Yellowed Santonin.—*Amer. Jl. Trop. Diseases & Preventive Med.* 1913. Aug. Vol. 1. No. 2. pp. 146-158.

The author in this paper gives an excellent account of sprue occurring in Porto Rico. In discussing the diagnosis of this

disease he insists on the presence of the following symptoms and signs:—

1. The typical sprue tongue.
2. The small liver.
3. The characteristic stool.
4. The long continued and excessive production of gas in the intestine.

These signs and symptoms are then discussed in detail. He then analyses 86 cases.

Firstly as regards race—

Americans	...	...	...	...	19
Porto Ricans	...	...	...	...	56
Spaniards	...	...	...	...	8
Natives of other Antillian Islands	...	...	...	...	2
German	...	...	...	...	1

Secondly as to age—

Under 20 (2 being 4 years old)	...	...	...	...	9
20-40	...	...	...	...	53
40-60	...	...	...	...	18
60-80	...	...	...	...	6

Thirdly as to circumstances—

11 were poor.

72 enjoyed the comforts of life to a greater or less extent.

Fourthly as to severity of symptoms—

Light cases	...	...	...	...	4
Moderate cases	...	...	...	...	30
Marked cases	...	...	...	...	39
Severe cases	...	...	...	...	10

Seventy-six cases were treated by the author, the treatment employed being:—

Milk "cure"	...	...	...	...	62
Meat	..	...	...	...	3
Fruit	..	...	...	...	1
General diet	...	...	...	...	2
On various diets + santonin	...	...	...	...	8

The results obtained were:—

Apparently cured	...	...	...	...	24
Improved	...	...	...	...	11
Unimproved	...	...	...	...	19
Result unknown	...	...	...	...	23
Died	...	...	...	...	9

The author then gives a detailed account of eight cases treated with various diets and santonin, and then of eight cases treated with various diets without santonin; the conclusions he draws from the study of these cases are that the santonin treatment does not seem to be superior to a carefully supervised diet, and that the most important factor in the dieting of these patients is to limit to very small amounts the starches, sugars and fats.

S. R. D.

## UNDULANT FEVER.

MOHLER (J. R.) & EICHORN (A.). **Malta Fever, with Special Reference to its Diagnosis and Control in Goats.**—*U.S. Dept. of Agriculture. 28th Ann. Rep. of Bureau of Animal Industry for the Year 1911.* pp. 119-136. With 4 plates. (1913. Washington: Govt. Printing Office.)

In this paper an interesting account of the history and etiological features of undulant fever is given; the authors point out that, contrary to the generally accepted view, the fever exists in the United States of America. After a careful investigation in the infected districts it appeared certain that under the names of "mountain fever" and "slow fever" the disease had existed for at least 25 years in Texas and New Mexico; that it always appeared among the people connected with goat raising, and that entire families have been taken sick with the fever on goat ranches. In some years the cases were numerous, in others few; they were most common after the kidding season, during the months of April, May, and June. Mexican goatherds are infrequently affected owing to the fact that they always boil the milk before drinking it. The disease is said to have prevailed in the country long before any improved breeds of goats were imported from South Africa; most of the goats in the infected region are of the Angora breed. The most important symptom observed in the infected goats was the frequency of abortions, which occurred in from 50-90 per cent. of pregnant females. In most cases the animals show no sign of sickness. The complement fixation test was found to be useful as a means of diagnosis where the agglutination tests gave doubtful reactions, and some very practical advice is given with regard to the preventive measures to be employed. It is stated that there is a tendency at the present time (1912) among physicians to advise the drinking of goats' milk for children and invalids; therefore the eradication of the diseased goats is very important from a public health standpoint.

P. W. Bassett-Smith.

TRIA (P.). **La Febbre Mediterranea.** (Note di Epidemiologia, Diagnostica, Profilassi.)—*Riforma Medica.* 1913. Apr. 5. Vol. 29. No. 14. pp. 380-383.

In this paper the author gives a short review of the epidemiology and the methods used for the diagnosis and prevention of undulant fever. Nothing new is brought forward, but the polymorphic character of the symptoms as found in the now widely extended endemic areas is specially emphasised, and the difficulty in differentiating some of the more irregular forms from paratyphoid and typhoid is noted. The complete and lasting immunity that can be produced artificially in animals is an important point; in dealing with goats in the affected regions an active immunisation is therefore possible. The reduction of the number of infected goats in Malta has been very great within recent years, but attention is drawn to the fact that in preparations made from



infected milk the *M. melitensis* can remain active for at least three weeks, and may therefore propagate the infection for that period.

P. W. B.-S.

**MEREU (F.). Febbre di Malta nella miniera Argentiera (Sassari).**  
[Undulant Fever in the Argentiera mine.] — *Policlinico*.  
Sez. prat. 1913. June 29. Vol. 20. No. 26. pp. 947-949.

MISSIROLI first described the presence of the disease in Sardinia. The author describes eight cases in which there was an irregular fever of long duration associated with enlargement of the spleen, sweats, articular pains and neuritis, which gave negative serum reaction with *B. typhosus* and positive with *M. melitensis*. He thinks that the diagnosis of these cases is very difficult and that many have been mistaken for malaria, typhoid and para-typhoid fevers. It is likely that the infection was derived from goats' milk as in the endemic area goats are present in great numbers and the unboiled milk was used by the sick.

P. W. B.-S.

**TROTTA (Guido). Ueber zwei Falle von Eiterung bei Maltafieber, welche Senkungsabszesse im Gefolge von Malum Potti vortäuschten.** [Two Cases of Suppuration in Undulant Fever, which simulated the Burrowing Abscesses of Pott's Disease.] — *Wein. Klin. Wochenschr.* 1913. Aug. 28. Vol. 26. No. 35. pp. 1395-1398.

In a septicaemic disease like undulant fever septic infections may follow, and recent research has proved that abscesses are not uncommon as sequelae. As complications the following have been described: Suppurative arthritis (GILMOUR & KENNEDY), Subphrenic abscess (EYRE & FAWCETT), Cholangitis (BULL), Endocarditis (BASSETT-SMITH), Pyonephritis (SCHOTTMÜLLER), and Pyonephrosis (CANTANI). The difficulties of diagnosis are often very great, as relapses of pyrexia are so common in this disease, but when the pus has collected surgical treatment becomes necessary. The two cases described by the author were in men of 45 and 52 years of age; both had contracted the fever about the same time and in the same district. Several months after, they complained of pains in the back and, later, swellings appeared in the crural regions. The clinical symptoms were like those of psoas abscess, slight fever was present, but there were no pulmonary signs or tubercular evidences. The serum of both reacted up to 1/200 with *M. melitensis*. No organisms were found in the pus and no sign of diseased bone was detected at the operations. Recovery was complete in both cases. The differential diagnosis had to be made between *M. melitensis* infection, Pott's disease, syphilis, and actinomycosis. The author states that he was guided mostly by the clinical characters, history, and serum reactions.

P. W. B.-S.

GARDNER-MEDWIN (F. M.). **A Case of Undulant Fever.**—*Liverpool Med. Chir. Jl.* 1913. July. No. 64. pp. 386-394. With 1 chart.

A description is given of a case of fever occurring in a male aged 64, who, while travelling in the Mediterranean for his health, apparently contracted undulant fever early in 1911 at one of the coast ports, probably in the south of France. The onset was very gradual but at Port Said he had marked arthritis, sweats, jaundice, and felt definitely ill. On arriving at Colombo he was landed and there CASTELLANI isolated the *M. melitensis* from his blood. After many relapses he returned to England in May 1912. The fever, associated with jaundice, continued to relapse from time to time and in January 1913 his condition showed high pyrexia, slight haemorrhages from mouth and lips, purpuric spots on the skin, and jaundice; the liver and spleen were both large. The urine contained traces of bile, and CAMMIDGE from an analysis of the faeces diagnosed catarrhal jaundice, probably of bacterial origin. From the urine an organism was isolated, believed to be the *M. melitensis*, and the patient's serum gave positive agglutination reactions with this, and the laboratory culture of *M. melitensis*, in dilution of 1/10 to 1/2000. Vaccine treatment was now commenced, and though the injections at first increased both the fever and the jaundice, obvious improvement set in, and in March he was able to do his work, but still was under vaccine treatment.

[The presence of jaundice is very rare in undulant fever. It is possible in this case that some intercurrent affection was the cause of the ill-health previous to the Mediterranean trip; but the very prolonged course, irregular symptoms, and the source of the infection suggest that the case may have been one due to a para-melitensis infection.]

P. W. B.-S.

SUAREZ DE FIGUEROA (D. José). **Cooperacion al Estudio de la Fiebre de Malta.**—*El Siglo Médico.* 1913. May 3. Vol. 60. No. 3,099. pp. 279-280; and June 14. No. 3,105. pp. 371-373.

These papers form a continuation of those of the author summarised in Vol. 1 of this *Bulletin* (p. 581). A clinical account is given of five cases in which the diagnosis was made bacteriologically.

P. W. B.-S.

VIGANÒ (L.). **Die Thermopräzipitinreaktion des Maltafiebers.**—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Aug. 4. Vol. 70. No. 34. pp. 200-202.

**Le Termoprecipitine del Micrococco Melitense.**—*Giorn. R. Soc. Ital. d'Igiene.* 1913. Aug. 31. Vol. 35. No. 8. pp. 337-340; and *Policlinico.* Sez. med. 1913. Sept. Vol. 20. No. 9. pp. 430-432.

The author has carried out a number of precipitin reactions for the diagnosis of undulant fever. He immunised a horse with

his strain of *M. melitensis* and during the process demonstrated from time to time that the serum possessed precipitating powers with a clear bacillary extract. He found that it was necessary to obtain a very clear extract; this had to be filtered through asbestos of a pure quality; if bad materials were used, pseudo-reactions were given. The bacillary extract was made from a 48 hour agar culture mixed with 25 c.c. of physiological salt solution and was treated either by heat in a water bath for 5 minutes, or by cold in the ice chest for 24 hours. The precipitating serum was obtained from infected animals which had been starved for 18 hours; and was made absolutely clear by centrifugation. When the test was carried out with the culture extract in a water bath or in the cold it gave at once, or quickly after, the opalescent ring reaction—at once with the first, more slowly and weakly with the second. Fifteen strains of *M. melitensis* were employed. With the heated extract clear sharp reactions were immediately obtained, the white ring becoming more and more strong during a quarter of an hour; though all were distinct, they were not all equally strong. Extracts made with staphylococci, streptococci, meningococci, gonococci, typhoid bacilli, &c., used as controls gave negative results. Extracts from the organs of guinea-pigs infected with *M. melitensis* were made in a similar way, reactions were given quickly and clearly with the extracts of spleen and liver, less strong with kidney extract, and not with heart extracts. To demonstrate the specific character of the reaction the following tests were made:—

1. Precipitating *M. melitensis* serum + extract of fresh material
2.     "                     "             " + extract of an animal infected with *M. melitensis*.
3.     "                     "             " + extract of *melitensis* culture.
4.     "                     "             " + extract of normal organs.
5.     "                     "             " + physiological salt solution.
6. Normal serum + extract of fresh material.
7.     "             " + extract of animal infected with *M. melitensis*.
8.     "             " + *melitensis* culture.

The normal serum was necessarily obtained from the same species of animal as the precipitating *melitensis* serum. These experiments appear to be worth following up, and are reliable if the material is well selected, and the technique carefully carried out. The author believes that he has established the reaction on a satisfactory basis by experimental work, and that in infections naturally acquired the process should be most useful in establishing a diagnosis of undulant fever.

P. W. B.-S.

#### ERRATUM.

*Mellin*, Vol. 1, p. 578, line 21 from bottom (end of first paragraph of BASSETT-SMITH's paper),—for "48 hours" read "two hours."

## HELMINTHIASIS.

## SCHISTOSOMIASIS.

BOUR (E. F.). Notes sur la Bilharziose.—*Bull. Soc. Méd. de l'Île Maurice*. 1913. Vol. 31. 2nd Ser. No. 32. p. 22.

In continuation of a communication made in December 1911 the author now recounts his attempts to infect experimentally the monkey, (*Macacus cynomolgus*), rabbit, and dog. The mouth, the nasal mucosa and the penis were experimented upon in the case of the monkey. The penis was first swathed in hot moist compresses for 45 minutes then totally immersed for an hour in water swarming with miricidia. The experiments upon the rabbit were as follows:—(a) Water containing about 50 miricidia was injected intraperitoneally. (b) Miricidia were applied for one hour to the skin of the ear which had previously been shaved and softened by hot compresses; and to granulations of a scarified ear. (c) Intravenous injection of very lively miricidia in a solution of one per cent. glucose in water.

In dogs the same experiments were made upon the penis, intravenous injections were also made.

These animals were under observation for a long time, the urine and faeces being frequently examined. The results, including those of a macroscopical and microscopical examination of the organs at the autopsy, were always negative. To explain this the suggestion of Professor LOOSS is adopted, viz., that man is the only host in which the bilharzia worm develops. [It should be stated however that the bilharzia worm found by COBBOLD in a monkey is a male *S. haematobium*].

R. T. Leiper.

MIYAGAWA (Y.). i. Ueber den Wanderungsweg des *Schistosomum japonicum* von der Haut bis zum Pfortadersystem und über die Körperkonstitution der jüngsten Würmer zur Zeit der Hautinvasion. [On the Route of *Schistosomum japonicum* from the Skin to the Portal System and on the Morphology of the young Worms at the Time of the Skin Invasion.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1912. Oct. 12. Vol. 66. No. 5/6. pp. 406-416.

ii. Ueber den Wanderungsweg des *Schistosomum japonicum* durch Vermittlung des Lymphgefäßsystems des Wirtes. [On the Migration of the *Schistosomum japonicum* through the Lymphatic System of its Host.]—*Ibid.* 1913. Mar. 1. Vol. 68. No. 2. pp. 204-206.

i. In this important memoir the cutaneous infection in bilharziosis hypothesized by LOOSS is proved by direct microscopical observation. The infective organism is elliptical, somewhat flattened, 0.04 mm. long and 0.015-0.022 mm. broad with various primitive organs. There is a thin body wall of double contour provided with short cilia and scarcely visible mouth, a rudiment of a ventral sucker, a primitive alimentary canal with peculiar, light brownish granular masses and no genital, muscular or nerve

tissues. The development of the oral sucker appears to Miyagawa to proceed much earlier than that of the ventral sucker which exists only as a tiny projection from the anterior third of the body.

The primitive gut is horseshoe-shaped, with the fore part closed and the posterior open, and ends in the anterior half of the body. Along this canal are peculiar light brown pigment granules probably identical with the pigment of the gut in the adult but finer. It was possible to identify the young worms discovered in blood from the peripheral veins with those from the portal blood. The same worms were found in the skin of the experimental animals. The young worms penetrate partly through the hair follicles, and in part directly through the healthy skin, and so enter not only the connective tissue spaces but also the blood capillaries. Thence they travel through the larger veins to the heart whence they subsequently reach the portal system. Whether they are carried in addition by the lymph stream is not yet clearly established but this is probable.

A comparison of these youngest worms with newly hatched miricidia reveals considerable differences and it is therefore concluded that *Schistosomum japonicum* most probably has an intermediate host.

ii. In the supplementary paper the additional fact is established that some of the infective agents of Bilharzia travel from the skin in the lymph stream. Miyagawa succeeded in recovering them from the thoracic duct and from lymphatic glands, but only in small numbers, in animals infected experimentally.

R. T. L.

TSUCHIYA. Clinical, Pathological-Anatomical, Pathogenic, Prophylactic and Therapeutic Study of the Schistosomiasis Japonica.—*Sei-i-kwai Med. Jl.* 1913. Aug. 10. Vol. 32. No. 8. Whole No. 378. pp. 107-109. [The original in No. 10. Vol. 27. 1913 of the *Jl. Tokyo Med. Assoc.*]

From studies made in the Yamanashi province of Japan in May and the succeeding months of the year 1910, the author arrived at the following conclusions.

Mild cases of Japanese schistosomiasis may show no clinical symptoms. Infected persons can then only be detected by the determination of the presence of eggs of *S. japonicum* in the faeces. These may however be absent in some cases of undoubted infection "Kabure," an inflammation of the skin which occurs in the province does not seem to have any direct relations with the site of entrance of the virus of schistosomiasis.

During the initial stages of Japanese schistosomiasis a rise of temperature and symptoms resembling typhoid or paratyphoid sometimes appear, but the general symptoms are slight, the serum reaction is negative, and during the course of the disease there is marked enlargement and tenderness of the liver, and enlargement of the spleen. The faeces contain eggs. When the fever lasts over a period, oedema and ascites frequently occur, and at times symptoms resembling those of dysentery are manifested.

The infectious period of the year appears to be from June till the end of October. No infection takes place during the cold months from November to April.

At times slight jaundice occurs in the later stages of the disease. In the last stage congestion of the portal circulation is the result not only of interstitial hepatitis but also, in severe cases, of endophlebitis and thrombus formation due to the irritation by the parasites producing haemorrhagic infarcts in the spleen and thrombosis in the portal system.

Infection invariably occurs through the skin. The parasites take seven to ten, rarely thirteen, days to reach the intrahepatic branches of the portal vein through the veins and lymphatic vessels. Once in the portal system they develop very rapidly and eggs are found in the faeces as early as one month after the date of infection while muco-haemorrhagic faeces are passed in 45 to 50 days.

The parasite seems to require most probably an intermediate host for its development, as all the worms hatched out of the eggs die within 40 hours, and all experiments to induce infection with them have failed absolutely. Attempts however to demonstrate an intermediate host experimentally have failed up to the present.

Prophylaxis consists, in the opinion of the author, in treating the cats and dogs that may act as reservoirs, with quinine. Faeces and urine should be stored so as to bring about the death of the eggs. FUJINAMI's methods of treating all dirty water with lime give good results where practicable. Individuals working in fields should be clothed and wear stockings and gloves made of closely woven material.

As treatment quinine in large doses and continuously is recommended.

R. T. L.

#### DISTOMIASIS.

SAMBUK (E.). *Distomatose pancréatique.*—*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. July. Vol. 4. No. 7. pp. 331-333.

The hitherto unobserved condition of infection of the pancreas by *Clonorchis sinensis* is now recorded. The patient in apparently good health died quite suddenly. At the post mortem a large number of flukes were found obstructing the bile ducts, and the duct of Wirsung and its branches. There was some indication of nephritis, but no other probable cause of death was noticed. The condition is considered very rare, as previous search of the pancreas in like infections has been negative.

R. T. L.

#### TAENIASIS.

NAUWERCK (C.). *Nochmals die "Durchbohrung des Duodenums und des Pankreas durch eine Tænie."* [Further Remarks on the Perforation of the Duodenum and Pancreas by a Taenia.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. June 21. Vol. 69. No. 5/6. pp. 434-436. With 2 coloured plates.

In 1900 STIEDA and NAUWERCK described a unique case of perforation of the duodenum and of the pancreas by a *Taenia*

*saginata*. Some doubts having been expressed by various writers as to the correctness of the interpretation of this case, the author now reproduces four photographs of the original preparations. [These illustrations leave no room for dubiety as to correctness of the original interpretation.]

R. T. L.

WATERHOUSE (R.). *Cysticercus cellulosae* in the Central Nervous System; with an Account of Two Cases.—*Quarterly Jl. of Medicine*. 1913. July. Vol. 6. No. 24. pp. 469-485. With 2 plates and text-figs.

Cerebral symptoms in an individual with cysticerci in the muscles or eye are probably attributable to the presence of similar parasites in the brain, especially if these symptoms comprise epileptiform convulsions, headache, giddiness, mental disturbance and vomiting. The occurrence of an eosinophilia in the cerebro-spinal fluid confirms the diagnosis. These cases otherwise are apt to be attributed to syphilis, or other cause, and should recovery occur whilst the patient is under specific treatment, the diagnosis is apt to be regarded as confirmed. Such errors would be generally prevented by the use of the Wassermann reaction. The clinical histories of two cases are given in detail and a series of instructive figures show the varying sites occupied by the wandering nodules or cysts at various periods.

The cerebral symptoms, which in one case commenced when the patient was the host of a tapeworm, attained their climax when the cysts in the muscles were at the height of their development, disappearing coincidently with their retrogression, to assume finally epileptiform convulsions which differed, as in other cases of the kind, from true epilepsy in the more gradual onset of the fits.

R. T. L.

BLANCHARD (R.). *Bertiella satyri*, de l'Orang-outang, est aussi parasite de l'Homme.—*Bull. de l'Acad. Méd., Paris*. 1913. April 15. Vol. 69. 3 sér. No. 14. pp. 286-296.

Several fragments of a tapeworm which were obtained from the stools of a girl of eight years of age from Port Louis in Mauritius by Dr. BARBEAU were sent to Professor Blanchard, and are described by him as belonging to the cestode, *Bertiella satyri*, hitherto known only from the Orang-outang of Borneo. There were six pieces amounting in all to 115 mm. and believed to belong to the same strobila. Four of these belong to the anterior portion of the worms and measure altogether 70 mm. The fifth portion is 30 mm. in length and 11 mm. broad at its anterior margin, the posterior being 12 mm. The last fragment 15 mm. long is 15 mm. across anteriorly and is rounded posteriorly in the manner which characterises the terminal segment of the members of the taeniidae. It is estimated that the complete worm would have measured at least 50 cms. in length and comprised several hundreds of segment.

The head is slightly elongated, and possesses neither rostellum nor hooks. It is not pigmented. The suckers are ovoid deep

and subcircular having a greatest diameter of 290  $\mu$ . The neck shows the first indication of segmentation about a millimetre behind the head. The segments increase in size very slowly and even the largest are still very short. The fourth fragment comprises 110 segments in a length of 28 mm., the greatest breadth being 4.5 mm. and the mean length 0.255 mm. There are 98 segments in the fifth fragment giving a mean length of 0.306 mm. with breadth varying from 11 to 12 mm. The terminal portion, 15 mm. by 15 mm., contains 26 segments, *i.e.* each has a mean length of 0.577 mm.

Whereas the segments do little more than double their length they increase in breadth more than four times.

The genital pores of the segment irregularly alternate, are small and difficult to see save in sexually mature segments. The cirrus pouch becomes evident about 20 mm. from the head. The uterus is transversely situated and shows on either side, *i.e.* anteriorly and posteriorly, a number of pouches or diverticula forming egg nests in the parenchyma. The eggs are round or oval and have three envelopes. The outer two are delicate; the innermost is thick, structureless, and transparent. At one of the poles the shell is thickened to form two little horns practically touching one another, with broad base and a short pointed tip. The diameter of this third shell is 20 to 25  $\mu$  and a thickness of 8 to 10  $\mu$  is attained where the horns are formed. The genus *Bertiella* (*Bertia*) is represented both in birds and mammals. The various species are given as follows:—

#### In Mammals.

In Primates.—*B. mucronata*, *B. cercopithecii*, *B. conferta*, *B. polyorchis*, *B. satyri*, *B. studeri*.

In Lemurs.—*B. elongata*, *B. plastica*.

In Rodents.—*B. americana*, *B. americana leporis*, *B. forcipata*, *B. laticephala*.

In Marsupials.—*B. edulis*, *B. obesa*, *B. rigida*, *B. sarasinorum*.

#### In Birds.

*B. delafondi* and *B. pinguis*.

The discovery of *B. satyri* in areas so widely separated as Mauritius and Borneo is no more remarkable than that of *Davainea madagascariensis* in Comores, Mauritius, Siam and British Guiana.

R. T. L.

#### ANKYLOSTOMIASIS.

MEIRA (Rubião) & PARANHOS (Ulysses). *L'Ankylostomiase au Brésil*. (Communication faite à la Société de Pathologie Comparée, dans la Séance du 11 février 1913.) 8 pp. [Imp. J. Thevenot, Saint-Dizier (Haute-Marne).]

This is an exhaustive summary of recent studies of ankylostomiasis in South America which are almost wholly inaccessible to European workers. The disease has been known in Brazil under the name "*Opilatio*" since 1845, and was first recognised



there as long ago as 1816 by HUMBOLDT. The earliest important Brazilian work however undoubtedly is that of JOBIM who proposed the name "intertropical hypoemia" for the disease in 1835.

The two ankylostomes are distinguished as "indigenous" and "imported." Out of 20,049 parasites collected from 110 patients 19,050 were *Necator americanus* and only 999 *Ankylostoma duodenale*. The two species were associated in no less than 38 out of these 110 cases.

It is stated that Gomes de FARIA and FEITOSA have infected themselves by applying necator larvae to the skin, and thus have confirmed for *Necator americanus* the original experiments made by Looss with *Ankylostoma duodenale*.

Dr. Marc DOWER has verified the existence of a haemolysin in the plasma of two out of seven cases of ankylostome anaemia, while Dr. O. PINTO has obtained a haemolytic action with *Necator americanus*. It would appear that, besides acting directly upon the red cells in the circulation, the haemolysin has some effect upon the haematopoietic organs.

ALMEIDA has noted that there is a degree of splenomegaly and hepatomegaly independent of, and much less marked than that occasioned by malarial infections. According to Meira there is in advanced stages an entire absence of free hydrochloric acid in the gastric juice, and the total acidity is considerably diminished. At the commencement of the illness, however, OLEVEIRA has observed a marked increase in chlorides, and in the total acidity. Blood is almost always present in the faeces but disappears after the patient is cured. The cardiac area is always increased, the right ventricle being especially liable to dilatation.

RABELLO gives as the characters of the blood: diminution of coagulability, of fibrin, of specific gravity, and of haemoglobin. A diminution of the red cells is not always noticeable; there may even be a polycythaemia due to diarrhoeic loss. Usually the haemoglobin diminishes to a greater degree than the red cells.

There is no leucocytosis. As a rule there is eosinophilia accompanied with a lowering of the neutrophiles.

The incidence of fever increases the proportion of neutrophiles and lowers that of the eosinophiles. In certain cases pseudo-eosinophile leucocytes are noted.

Usually a variable meta-basophilia occurs but only to a slight degree. The nucleated red cells occasionally seen are almost always normoblasts. When megaloblasts are present, they are fewer in number.

Dr. P. da CUNHA has studied the jaundice type of ankylostomiasis, rare in Brazil, in which typical jaundice is associated with yellow pigmentation of the sclerotics, with biliary pigment in the blood and a total absence of symptoms of biliary intoxication. These cases are characterised by anaemia, globular fragility and granular red cells. There is a constant disproportion between the greatly diminished haemoglobin and the increased number of the red cells; leukocytosis, polychromatophilia and a strong hemopoietic reaction (nucleated red cells and myelocytes) are absent.

The mental perturbations associated with ankylostome infections are the subject of a monograph by Professor AUSTRAGESILO and Dr. H. GORRIZZO of Rio, to which attention is directed.

It is the view of the authors that ankylostomiasis has not attained a grave character in Brazil. It is so widespread that had it been a fatal disease certain regions would soon have been decimated. Cachexia rarely occurs, and few of the sick reach the last stage without seeking treatment. Treatment adopted at Sao Paulo is thymol in 6 gram doses, taken fasting, in capsules of 1 gm. every fifteen minutes followed by a purgative. An iron tonic is given afterwards. The treatment may be repeated eight days after the administration of the first dose of thymol. This is persisted in until the faeces no longer contain eggs. The authors have never experienced the accidents attributable to thymol. The drug is always well tolerated but the dosage should be varied according to the age of the patient, the degree of anaemia, etc. The worms are expelled by hundreds and cases are not rare in which a thousand worms have been removed by a single dose.

Nothing in the direction of prophylaxis has yet been undertaken by the public authorities in Brazil but it is recognised that if the spread of the disease is to be controlled there is urgent need for state intervention.

R. T. L.

CONRAN (P. C.). **A Report on Ankylostomiasis in the North Nyasa District.**—*Jl. Trop. Med. & Hyg.* 1913. July 1. Vol. 16. No. 13. pp. 195-198; and *Nyasaland Protectorate Ann. Med. Report on Health and Sanitary Condition for Year ended 31st March, 1913.* pp. 68-72.

During a stay from June to December of 1912 in North Nyasa the author examined the stools of 622 individuals and obtained the following interesting results in Karonga and adjoining villages:—

Ova.	No. of infected persons.	Per-centage.	Men per-centage.	Women per-centage.	Boys per-centage.	Girls per-centage.	Infants per-centage.
Ankylostomum ...	211	40.42	44.0	42.67	30.0	32.35	45.83
Schistosomum ...	169	32.38	31.0	16.0	48.57	44.12	29.17
Ascaris ...	47	9.0	5.0	11.33	1.43	8.82	22.92
Trichocephalus ...	8	1.54	1.0	1.33	1.43	2.94	2.08
Negative ...	87	16.66	—	—	—	—	—
Total ...	522	—	—	—	—	—	—
At Fort Hill (Mambwe tribe in scattered mountain villages):—							
Ankylostomum ...	13	—	15.79	16.22	12.0	5.26	—
Schistosomum ...	11	—	10.53	8.11	8.0	21.03	—
Ascaris ...	7	—	5.26	5.41	8.0	10.53	—
Trichocephalus ...	2	—	—	—	4.0	5.26	—
Negative ...	67	—	—	—	—	—	—
Total ...	100	—	—	—	—	—	—

The author remarks that the extent of the disease amongst children in the Karonga district makes itself evident in the exceedingly high infant mortality, disease from which a healthy child would easily recover proving fatal owing to lack of resisting power. He notes that the districts (Mwangolera and Mwambungo) with the highest percentage of ankylostome infections lie inland at some distance from the shore of the lake and derive their water supply from the upper Rukuru and its tributaries or from shallow pools: the districts of Chisindiri and Kambombo on the other hand are near the mouth of the river Rukuru and obtain their water supply from the Lake or the wide mouth of the river. [This contrast is not so evident in the incidence of schistosoma infection as will be seen from the appended table]:—

Ova.	Chisindiri.	Kambombo.	Mwangolera.	Mwambungo.
Ankylostomum ...	41·6	25·4	43·4	50·9
Schistosomum ...	31·6	38·1	38·5	38·18
Ascaris ...	3·3	6·3	13·2	10·9
Trichocephalus ...	—	—	2·4	—

Of the total number of persons examined in the Karonga district 28·54 per cent. had ankylostomes alone, 18·77 per cent. had schistosomes while in 11·85 per cent. only did the two infections occur together.

Most of the symptoms commonly described were met with but particularly emphasized were (most constantly) pain and tenderness in the epigastrium, dyspnoea, palpitation, weakness, dizziness, headache. Joint pains simulating rheumatism were common. Although many patients complained of passing blood with the motions, melaena was never observed in pure ankylostome infection but was always associated with schistosomes. In these cases there may be a close resemblance to dysentery, almost pure blood being passed with mucus, and accompanied with marked griping and tenesmus. The paper concludes with an interesting account of the local conditions with special reference to the possibility of enforcing prophylactic measures through the authority of village headmen.

R. T. L.

WYLER (E. J.). Some Observations on Ankylostoma Infection in the Udi District of the Central Province, Southern Nigeria.—*Jl. Trop. Med. & Hyg.* 1913. July 1. Vol. 16. No. 13. pp. 193-195; and *Southern Nigeria Ann. Med. Report for Year ending December 31st, 1912.* pp. 40-45. [1913. London: Printed by Waterlow & Sons, Ltd.]

This report [which appears in essentially the same form in the two publications above] deals with the results of an examination of the stools of 200 persons incarcerated in the Udi jail over a period of eight consecutive months. The individuals

came originally from over 65 villages scattered widely throughout the district approximately 1,537 square miles in extent, and are held to be representative of the general populace which numbers approximately 460,000. It is stated that hookworm infection is practically universal (199 positives out of 200) the average individual infection is a heavy one. Both species *A. duodenale* and *N. americanus* occur. Pure infections unmixed with other parasites are found to the extent of about 44 per cent. of the cases; in 12½ per cent. *Ascaris lumbricoides* is associated; and in 29 per cent. *Trichocephalus trichiura*. 79 per cent. of the population is anaemic but the anaemia is rarely profound. The pulse rate is normal in about half of those examined. Ground itch on the feet has not been observed. The people of the district exhibit generally a poor standard of physique, they have a marked incapacity as carriers and workers, and an obvious disinclination for bodily exertion in contrast to the Mendies, Kroo, Fantis, Yomba, Hausa and other coast tribes. This is probably attributable to the saturation of the inhabitants of the Udi district with ankylostome infection. [This paper acquires some importance from the fact that Udi promises to become a great coal centre. The construction of an important railway has just been sanctioned.]

R. T. L.

**BREINL (A.). Report on Health and Disease in the Northern Territory.—Bull. of the Northern Territory. 1912. July. No. 1a. p. 10.**

Ankylostomiasis has so far been observed in the Northern Territory of Australia in a few cases only. Three in 1908 and one in 1910. It was not possible to determine whether this disease was contracted locally, or whether the patients had brought the infection with them from other regions of the Commonwealth.

R. T. L.

i. **DINSMORE (W. W.). Hookworm Disease, a National Problem.—Southern Med. Jl. 1913. Aug. Vol. 6. No. 8. pp. 498-506. With 8 illustrations.**

ii. **BOERNER (M. H.). The Hookworm Problem: Synopsis of the Work of the Hookworm Commission of the Texas State Board of Health.—Texas State Jl. of Med. 1913. Aug. Vol. 9. No. 4. pp. 133-134.**

iii. **JUDKINS (O. H.). Symptoms and Diagnosis of Hookworm Disease.—Ibid. pp. 134-136.**

These papers belong to the epoch-making chapter in the vast literature of ankylostomiasis which deals with the propaganda of STILES and Wickcliffe ROSE, the leaders of the Rockefeller Commission for the Eradication of Hookworm. The methods adopted have already been given in detail in the annotation

upon the official publications of the Commission (see this *Bulletin*, Vol. 2, p. 182).

i. Dinsmore's paper is of a popular character and is strikingly illustrated.

ii. Boerner states that 20,418 persons of all ages and both sexes have been examined at the special dispensaries during the nine months since August, 1912, and 8,892 (*i.e.* 43.5 per cent.) were found infected. The most heavily and most frequently infected class was the school child. Of children between six and eight years of age 5,775 out of 9,649 (*i.e.* 59.8 per cent.) were infected, while of 562 negroes only 57 or 10.1 per cent. gave positive results.

iii. Judkins, reviewing symptoms and diagnostic features, divides the infections into mild, medium, and severe. The first class contains many patients who present no symptoms recognisable as variations from the normal, yet after being treated, these often gain from five to ten pounds in weight and feel much better than before. In such cases mild digestive disturbances and discomfort in the epigastrium, presumed by the patient to be normal, are to be regarded as due to this cause. Many show also moderate pallor, lowered vitality and diminished energy. Children grow less rapidly and are not so bright mentally. The appetite is apt to be capricious, and attacks of gastralgia with occasional meteorism are noticeable.

In the *medium* class all these conditions are accentuated. The skin has a peculiar pasty appearance, and the mucous membranes are paler. The appetite is exaggerated or perverted; the tongue is coated and flabby, and there is epigastric tenderness. Palpitation is readily induced; there is mental depression and often stupidity. The haemoglobin in these cases ranges from 30 to 60 per cent.

*Severe* cases show extreme pallor, generally oedema, severe digestive disturbances, appetite enormous or absent, diarrhoea alternating with constipation, nausea, vomiting, and ascites. The patient is melancholic and complains of frequent headaches. There is extreme weakness, irregular fever and dyspnoea, severe palpitation and pericardiac pain. Heavily infected children are often "pot-bellied" due to dilatation of the stomach and intestines, and to increase in the mesenteric fat.

Acute infections often simulate typhoid fever or malaria.

R. T. L.

**STILES (Ch. Wardell) & BOATWRIGHT (Hal. F.). Thymol Administration. Subjective Effects in 464 Administrations in 243 Patients.—U.S. Public Health Reports. 1913. July 18. Vol. 28. No. 29. pp. 1497-1513.**

The authors give in full details an important series of "subjective symptoms" reported by patients treated, from one to seven times each, according to circumstances, with the following "stock treatment." One or two preliminary doses of Epsom salts (at

5 and 8 p.m. or in the evening of two succeeding days), next morning thymol (divided into two doses at 6 and 8 o'clock or in three doses at 6, 7, and 8 o'clock) followed by salts at 10 o'clock. The results are summarised in their order of frequency in the following table:—

Symptoms, etc.	Treatments.	
	Number.	Per cent of total.
Total number of patients, 243.		
Total treatments ... ..	464	100·0
Ill effect absent ... ..	259	55·8
Ill effect present (as follows) ... ..	205	44·1
Sickness at the stomach (nausea) ... ..	66	14·2
Weakness ... ..	62	13·3
Burning in the stomach ... ..	45	9·7
Dizziness (including also "giddiness," "drunk," "staggy") ... ..	44	9·4
Headache ... ..	14	3·0
Vomiting ... ..	13	2·8
Burning in the throat ... ..	8	1·7
Pain in the stomach* ... ..	7	1·5
Drowsiness or sleepiness... ..	5	1·1
Sick after discharge from treatment ... ..	3	·64
Chill (apparently not due to treatment) ... ..	1	·21
Dyspnea ... ..	1	·21
Irregular heart following thymol ... ..	1	·21
Fainted ... ..	1	·21
Deaths ... ..	0	0·

\* The term "stomach" used by patients does not necessarily correspond exactly with the anatomical term.

They remark that the most common complaint, *nausea*, was certainly due in some instances to the thymol, but in others it was attributable to the mag. sulphate.

The "weakness" noticed in 13·3 per cent., was at times due apparently to thymol or salts, but in other cases to the fact that the patients were allowed no breakfast. The number of complaints has been reduced by allowing coffee without milk at 9.30 a.m. and again, with crackers but no butter at 10.30 a.m. This is only partly efficacious however.

Some patients developed symptoms only at later treatments, others who showed ill effects at the first, had none subsequently. An idiosyncrasy exhibited at the first treatment is not a necessary criterion of what may follow subsequently. Generally patients feel better when they remain in bed as much as possible until dinner time, but some cases have felt no ill effects when they have not done so. They conclude by urging that thymol and salts alone will not suffice to eradicate hookworm disease. The sanitary privy [and what it represents] is also essential.

ARCHIBALD (R. G.). A Case of Acute Agchylostomiasis treated by an Autogenous Vaccine of a Coliform Organism.—*Jl. Trop. Med. & Hyg.* 1913. Sept. 1. Vol. 16. No. 17. pp. 260-262.

In a certain class of cases of ankylostomiasis the signs and symptomatology are out of all proportion to the number of worms obtained from the host. In the opinion of the author this is probably due to the presence of an additional toxic agent in the intestinal tract. The varying types of fever which sometimes occur and the persistence of pyrexia after the expulsion of the worms are difficult to explain on the supposition that the ankylostome is the sole causal agent. Processes of intestinal putrefaction are very marked in this disease. The author considers that the resultant toxins must necessarily affect the health of the patient, and makes a plea for the trial of vaccine therapy as a means, in serious cases, of tiding the patient over a "toxaemic time", previous to the exhibition of the anthelmintic, which being itself of a toxic nature, might have an injurious effect. The case cited gives strong support to the view that beneficial results can be obtained in severe and intractable ankylostomiasis by the employment of autogenous vaccines of the suspected intestinal organism along with, or prior to, specific anthelmintic treatment. The faeces were very offensive and alcoholic, and contained large numbers of adult ankylostome worms and their ova. Eucalyptus and chloroform in large doses produced little or no benefit and a fatal issue seemed imminent. An almost pure culture of a coliform organism was obtained by suitably plating out a specimen of faeces, and a vaccine of the organism was prepared. An injection of 500 million organisms was followed by immediate improvement. The tongue became clean and all the toxaemic signs disappeared. Five days later a dose of 1,000 million was used, and this was repeated subsequently at the end of a week. After the third injection the temperature remained consistently lower, the spleen and liver diminished in size, the patient commenced to put on weight rapidly, and was discharged from hospital shortly afterwards. The patient continued in good health and was able to carry on manual labour, although ankylostome ova were still present in the faeces.

[It may be well to point out here that the use of the spelling *Agchylostomiasis* is pedantic and has no justification in the International Rules of Nomenclature. These rules apply solely to Nomenclature not to Terminology. Thus, though we may consider it necessary, as some do, to use the generic word *Agchylostoma*, the fact does not invalidate the use of "Ankylostomes" and "Ankylostomiasis" in a descriptive sense.]

R. T. L.

#### ASCARIASIS.

LAMOUREUX (A.). Fréquence du Parasitisme intestinal par *Ascaris lumbricoides* et par *Trichocephalus trichiuris* chez les Habitants de la Grande Comore.—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 455-457.

On account of the death from ascariasis of a young soldier from the island of Grand Comore the stools of 41 Comore individuals recently brought to Majunga in Madagascar were

examined. No less than 39, i.e. 95.1 per cent. were found infected by the *Ascaris lumbricoides* and *Trichocephalus trichiuris*; in association in 73.17 per cent.; by ascaris alone in 19.51 per cent.; and by trichocephalus only in 2.43 per cent. The high percentage in people coming from Grande Comore led to an examination of Madagascar natives living under the same conditions of life and food in Majunga. 31 artillery men gave only 3 (9.6 per cent.) infections:—two by ascaris alone and one with ascaris and trichocephalus in association.

In a foot-note it is stated that the infected inhabitants of Comore were feverish and had gastro-intestinal trouble. These fevers are frequently subject to relapse. No malaria parasites or spirochaetes were found in the blood. This fever is undoubtedly due to intestinal parasites and gives way to anthelmintic treatment. This remark is of importance in malarial countries where these relapsing fevers, diagnosed without blood examination, are frequently confounded with malaria.

R. T. L.

ALLEN (Mary D.). *Ascaris lumbricoides* as a Complication of a Surgical Operation.—*Jl. Amer. Med. Assoc.* 1913. June 21. Vol. 60. No. 25. pp. 1953-1954.

A remarkable case is recounted in which, after the evacuation of a large abdominal abscess a number of *Ascaris lumbricoides* appeared at intervals in the dressings and delayed the healing of the wound over a period of four months.

R. T. L.

#### GENERAL AND UNCLASSIFIED.

TENNEY (Elmer S.). Some Observations on the Prevalence of Intestinal Parasites in the Philippine Islands.—*Amer. Jl. Trop. Diseases & Preventive Med.* 1913. July. Vol. 1. No. 1. pp. 44-48.

In 1912 the author found from an examination of the stools of a company of 100 Philippine scouts that *Ascaris lumbricoides* occurred in 52, *Trichuris trichiura* in 48, and "Uncinaria" in 24 cases. Since then a further series of 150 persons (comprising 121 Philipinos, 27 Moros, and 2 Chinese) examined has given the following results:—

			per cent.
<i>Trichuris trichiura</i>	...	67	44.6
"Uncinaria"	...	50	33
<i>Ascaris lumbricoides</i>	...	48	32
<i>Strongyloides stercoralis</i>	...	5	3.3
<i>Dibothriocephalus latus</i>	...	1	0.6

The term "Uncinaria" is used to include *A. duodenale* and *N. americanus* as the differentiation of these from the egg was impracticable.



An examination of the stools of 21 American residents, including officers of the army and of the Philippine Constabulary as well as several women and children, who had been in the Islands for over a year resulted in the following figures:—

	per cent.
<i>Trichuris trichiura</i> ... ..	9.5
<i>Ascaris lumbricoides</i> ... ..	9.5
" <i>Uncinaria</i> " ... ..	4.7
<i>Oxyuris vermicularis</i> ... ..	4.7
<i>Strongyloides stercoralis</i> ... ..	4.7

These results should be compared with those of CHAMBERLAIN, BLOOMBERGH and KILBOURNE (*Philippine Jl. of Science*, B., 1910, Vol. 5, No. 5), and STILES and GARRISON (*U.S. Hygienic Laboratory Bulletin* No. 28, 1906).

R. T. L.

SALECKER. Über Helmintheninfektionen bei den Eingeborenen der Marianen. [Helminthic Diseases in the Ladrone Islands.]—*Arch. f. Schiffs- u. Trop. Hyg.* 1913. July. Vol. 17. No. 13. p. 463.

An examination of the stools of about 10 per cent. of the population of the island of Rota gave the following:—

Ankylostomiasis ... ..	83 per cent.
<i>Ascaris</i> ... ..	93 „ „
<i>Trichocephalus</i> ... ..	89 „ „

Many of the ankylostome carriers were very anaemic. In remarks upon the ankylostomiasis campaign in Guam the importance of a periodical examination is emphasized. In spite of a thorough course of treatment the children were found freshly infected some months later.

R. T. L.

BEDSON (S. Phillips). Lésions des Organes à Sécrétion interne dans l'Intoxication Vermineuse. — *Ann. Inst. Pasteur.* 1913. Aug. 25. Vol. 27. No. 8. pp. 682-699. With 6 text-figs.

From observations made upon guinea-pigs injected with sterile peri-enteric fluid from *Ascaris megalocephala* and filtered extracts of various tapeworms (*T. plicata*, *T. perfoliata* and *T. saginata*) the following conclusions are drawn.

Of all the organs of internal secretion the suprarenal capsule presents the most important lesions in acute or chronic verminous intoxications. The thyroid gland reacts equally but more especially in subacute and chronic cases. Only slight lesions occur in the hypophysis, pancreas, ovary and testicles.

The severity of the lesions in the suprarenals and thyroid is dependent more on the number of the injections and the duration

of the intoxication than upon the amount of toxin injected. Verminous products of ascarids and taenia produce identical lesions.

The reaction of the experimental animal to the verminous toxin is specific. In some cases the suprarenal capsule reacts quickly at the first injection of toxin, and even may present indications of hyperfunction. On the other hand the suprarenals may continue to show very little reaction.

The thyroid as a rule reacts but feebly to acute intoxication, but after intoxication of some weeks' duration extensive sclerosis obtains. It is stated in conclusion that the organs of internal secretion react quite similarly to verminous toxins as to those of microbic origin or to injurious chemical substances.

R. T. L.

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## FILARIASIS.

BREINL (Anton). On Human Filariasis in Queensland and the Morphology of *Microfilaria Bancrofti*.—*Australian Institute of Tropical Medicine. Report for the Year 1911.* pp. 18-23.

According to the author's short experience filarial infection in man is not uncommonly met with in Northern Queensland, but it is apparently not as prevalent as in and around Brisbane. A systematic examination of the patients who entered Townsville Hospital during three months showed that of 164 men examined five harboured embryos of *Filaria bancrofti* in their blood and of 60 female patients, two. The embryos evinced a well marked periodicity, being absent from the blood in the day and appearing at night. The author details some work on their morphology this being similar to that described by various workers in different parts of the world. As regards the pathology of the disease, in three cases of filarial swelling the varicose groin glands and the hyperplastic lymph vessels were removed and histologically examined. Only in one case, in the enlarged spermatic lymph vessels, could live adult female *F. bancrofti* be found. These agreed in every respect with the measurements and previous descriptions of this parasite.

On the histological examination of the enlarged lymph glands no marked pathological changes were evident. "The tissue, which caused the "soft swelling," consisted, on the whole, of loose, richly vascular fibrous tissue containing lymphocytic infiltration. Here and there, in parts which showed a greater amount of small celled infiltration, lymphocytes had collected in round or irregular follicle-like patches from 200 to 800 $\mu$  in diameter. These lymph follicles occurred in varying numbers, were well defined from the surrounding tissue, and richly vasculated. As a rule, two areas could be distinguished, a peripheral one, consisting of lymphocytes, and a central one, consisting of more lightly staining cells, resembling the epithelioid cells of a tubercle. In all sections, especially in those of the lymph tissue surrounding the spermatic cord, numerous larger and smaller lymph vessels could be seen. The larger ones had a very thick wall and a wide lumen. Many of the larger lymph vessels, however, showed thrombus formation, the lumen of the vessel being filled by a fine loose meshwork of newly-formed connective tissue, and often nearly obliterated."

"Throughout the connective tissue haemorrhagic infiltration to a varying extent could be noticed. In some parts haemorrhage had taken place, the blood was being reabsorbed, becoming organised by connective tissue fibres growing in from the surrounding parts."

Only in one case was a dead adult *F. bancrofti* met with; it was seen on cross sections, surrounded by a thick layer of dense fibrous tissue.

A coloured plate illustrates the paper.

G. C. Low.

**NYASALAND PROTECTORATE. Annual Medical Report on the Health and Sanitary Condition for the Year ended 31st March, 1913. p. 10.**

Filariasis is rarely reported by Medical Officers in Nyasaland, elephantiasis of the legs or scrotum being seldom observed. It is, however, evident that in certain districts the existence of the infection may be readily demonstrated if the parasite is specially searched for. SANDERSON in the Ruo district, for example, examined 177 natives, blood films being taken during the day from 66, and from the remainder after 8 p.m. and was rewarded by finding embryonic forms of *F. bancrofti*. "Of the 66 natives examined between 8.30 a.m. and 4.30 p.m. positive results were obtained in five, or 7.5 per cent. Of the 111 examined after 8 p.m., filariæ were found in as many as 31, which gives a percentage of 27.92. In none of the cases which yielded a positive result were there any signs of elephantiasis present." Only the one species of filaria, namely *F. bancrofti*, was found.

[The absence of *F. perstans* and *F. loa* in the 177 natives examined is interesting.]

G. C. L.

**SALM (J.). Un Cas de Filariose observé dans les Iles de la Sonde.—*Le Caducée*. 1913. August 2. Vol. 13. No. 15. pp. 205. With 1 fig.**

According to the author cases of filariasis in the Dutch Colonies are rare. The case of a man suffering from varicose groin glands is described, and an excellent photograph shows very distinctly the lesions that were present. These consisted of irregular soft tumour masses in the left groin and upper part of the left thigh. Embryo filariæ were found in the night blood in small numbers, never in the urine nor in the fluid aspirated from the swellings. A marked eosinophilia was present, in one examination as high as 35 per cent.

G. C. L.

**MAILLE. Deux Cas de Filariose du Sang. Étude Hématologique.—*Arch. de Méd. et Pharmacie Navales*. 1913. June. Vol. 99. No. 6. pp. 462-466.**

Two cases of *Filaria loa* infection are described, in one of these Wassermann's reaction was obtained, this evidently meaning an infection with syphilis as well.

In the first case numerous filarial embryos were present in the blood. [The statement that an adult *Filaria loa* male was found in the blood is manifestly incorrect.] The second case was a typical one of Calabar swellings. The patient, a Frenchman, had been twice on the Congo and only developed the swellings on his return home.

G. C. L.

**AUBERT (P.) & HECKENROTH (F.). Action de Divers Médicaments sur les *Microfilaria perstans* et *diurna*.—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 457-459.**

The authors first refer to the interesting results reported by THIROUX, D'ANFREVILLE and LEMOINE on the action of aniline

tartar emetic (*émétique d'aniline*) in filariasis. The authors tried the drug and various arsenicals on cases infected with *Filaria loa* and *F. perstans*. They found that as regards atoxyl, arsenophenylglycin, and "606" the results were entirely negative, while as regards the aniline tartar emetic they could not honestly say that it had any definite therapeutic action on the filaria. They call attention to the variability of the microfilariae in the peripheral blood, and rightly point out that unless this is taken into account wrong interpretations may be given to the action of drugs.

G. C. L.

v. d. HELLEN. **Arsenpräparate und Filarien.** [Arsenic Preparations and Filariasis.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 13. p. 462.

Atoxyl and arsenophenylglycin were tried in the treatment of filariasis, but were found to be useless. No details are given.

G. C. L.

BACH (Fritz Werner). Ueber die "Mikrofilarienkulturen" von Wellman u. Johns, nebst Bemerkungen über die Messung der Mikrofilarien. [On the Microfilaria Cultures of Wellman and Johns with Observations on the Measuring of Microfilariae.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. July 29. Vol. 70. No. 1-2. pp. 50-60. With 1 plate.

The author repeated the experiments of WELLMAN and JOHNS [see this *Bulletin*, Vol. 1, p. 91] using the same species of filaria, namely *Dirofilaria immitis*.

In none of his cultures did he notice any appreciable increase in length of any of the embryos. In some, however, a slight increase of  $20\mu$  was obtained, but as this was also seen in controls in blood, to which nothing had been added, it cannot be taken as meaning anything. He cannot therefore confirm WELLMAN and JOHNS's results.

He mentions and agrees with Low's criticisms of the same paper [see this *Bulletin*, Vol. 1, pp. 91 and 420], where it was pointed out that the first stage of the development of the embryo filariae is one of breadth and not of length.

The second part of the paper is devoted to the longitudinal measurements of embryo filariae, their size when dead and when in the vagina of the adult female and the question of their agglomeration by sera.

[The paper contains many points of interest and shows evidence of very careful and accurate work.]

G. C. L.

#### FILARIAL ABSCESS.

ZIEMANN (H.). Beitrag zur Lehre tropischer Gewebsentzündungen infolge von Filariainfektion. [Contribution to the Knowledge of Connective Tissue Inflammations in the Tropics, due to Filarial Infection.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 14. pp. 469-493.

The author states that there exists in New Guinea a very characteristic form of connective tissue inflammation or purulent

suppuration, presumably due to filariae and found chiefly in the musculature. He pointed this out in the year 1904 and described the condition as a disease *sui generis*. He did so for several reasons:—

- (1) On account of the tendency for the foci, especially in the lower extremities, to be multiple.
- (2) On account of the successful results of surgical interference.
- (3) On account of the frequent sterility of the contents of the abscesses.
- (4) On account of the frequent absence of fever.
- (5) On account of the majority being deeply situated and therefore in the beginning correspondingly difficult to diagnose.
- (6) On account of the frequent absence of definite suppuration, the pathological lesions consisting of serous infiltration with small celled infiltration.

In many of the abscesses secondary infections with staphylococci and streptococci were found, but the author considers the primary etiological agent to be the filaria. He thinks the suppuration might be due to the death of the mother worm, or to an abortion of its eggs or perhaps partially to toxic products proceeding from the worm.

The condition was generally found in people with larval filariae in the blood, or in the absence of those a marked eosinophilia indicated the filarial nature.

As regards treatment the author advises waiting, if the pain is not too great and pus has not formed. Frequent blood examinations and careful records of the temperature should of course be kept. When pus forms the abscesses should be freely opened and treated on surgical lines.

The prophylaxis lies in the destruction of the Culicidae which act as intermediate hosts of the filariae.

[Similar cases have been referred to lately in this *Bulletin*, see Vol. 2, pp. 92-96.]

G. C. L.

KÜLZ (L.) & BACH (Fr. W.). *Beiträge zur Kenntnis von Onchocerca volvulus* Leuck. 1893. [Observations on *Onchocerca volvulus* Leuck.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Aug. 23. Vol. 70. No. 5-6. pp. 321-326. With 6 text-figs.

The specimens were recovered from tropical muscle abscesses by SCHÄFER in the Cameroons. They were sent to the authors from the Zoological Museum at Berlin. The worms were preserved in alcohol and were stained a light brown colour; they were coiled up together and in unravelling them many got broken. An examination showed them to be *Onchocerca volvulus* = *Filaria volvulus*, this species having been placed in the genus *Onchocerca* instead of in that of *Filaria* by RALLIET and HENRY in 1910. Amongst the fragments two heads, two tails, and four headless and tailless bodies were found. Sketches of these are given together with measurements of the different anatomical

points, these being compared with those given by PENEL, PROUT, FULLEBORN, BRUMPT and PARSONS.

No clinical details were obtainable about the patients who had had the worms, but the authors point out that this parasite usually produces fibrous tumours. They also mention the frequency with which muscle abscesses occur in the Cameroons, but, from epidemiological and clinical researches, incline to the view that these are due to *Filaria loa* [see this *Bulletin*, Vol. 2, p. 92].

G. C. L.

**BREINL (Anton).** Investigation into the Morphology and Life History of *Onchocerca gibsoni*.—*Australian Inst. Trop. Med. Report for the Year 1911*. pp. 5-17.

The observations given in this paper are of medical interest mainly as bearing upon the life history of the closely allied parasite of man *Onchocerca volvulus*.

Previous work by GILRUTH and SWEEER had indicated the possibility that a tick was the intermediary host while CLELAND, on epidemiological grounds was led to suspect the stomoxys.

The author states the curious fact, which is regarded as more than a coincidence, that the nodules occur only in those parts of the animal which come into contact with the ground when the beast is resting, or in the water, when the cattle enter it for drinking or cooling purposes. It seems possible therefore that larvae may be able to penetrate the unbroken skin and thus gain access to water and a water-living intermediary. [It may be recalled however that *O. reticulata* is differently situated in the horse]. A large number of experiments were made which appear to show that in a few instances such a migration of embryos in small numbers does take place when the skin over nodules is kept moist, but it is concluded that this migration is of too rare an occurrence to be taken into consideration. It may be merely a pathological curiosity. Experiments made with *Stomoxys calcitrans* [c.f. LEIPER, *Bulletin*, Vol. 2, p. 196.] *Culex fatigans*, *Culicella vigilax*, *Mansonia uniformis*, *Hirudo medicinalis* and *Cyclops pallidus* n. sp. were entirely negative. Those upon the last named are apparently conclusive for although the larvae were repeatedly taken up by the Cyclops, no development resulted and after three to four days the worms were digested.

R. T. Leiper.

#### ELPHANTIASIS

**MATAS (Rudolph).** The Surgical Treatment of Elephantiasis and Elephantoid States dependent upon Chronic Obstruction of the Lymphatic and Venous Channels.—*Amer. Jl. of Trop. Diseases & Preventive Medicine*. 1913. July. Vol. 1. No. 1. pp. 60-84.

The author reviews some of the most recent suggestions which have been offered for the relief of elephantiasis and elephantoid states dependent upon chronic obstructions of the lymphatic and venous channels. He also describes the results of two recent cases operated upon by himself by the method of KONDOLRON of Athens.

By elephantiasis he means "a progressive histo-pathologic state or condition which is characterised by a chronic inflammatory fibromatosis or hypertrophy of the hypodermal and dermal connective tissue, which is preceded by and associated with lymphatic and venous stasis, and may be caused by any obstruction or mechanical interference with the return flow of the lymphatic and venous currents in the affected parts. In order to bring about the hypertrophy of the connective tissue, which is the distinctive feature of the true elephantiasis state, the mechanical impediment to the lymphatic and venous drainage of the part is not sufficient, because a simple mechanical obstacle, while causing a regional or localised dropsy or lymphoedema, will not bring about the characteristic fibromatosis and other histological changes which are peculiar to elephantiasis."

The histopathological elements which are essential to complete the picture of elephantiasis are according to Matas:—" (1) a mechanical obstruction or blockage of the veins and lymphatics of the region, usually an obliterative thrombophlebitis or lymphangitis or adenitis; (2) hyperplasia of the collagenous connective tissue of the hypoderm; (3) gradual disappearance of the elastic fibres of the skin; (4) the existence of a coagulable dropsy or hard lymph oedema; and (5) a chronic reticular lymphangitis caused by secondary and repeated invasion of pathogenic micro-organisms of the streptococcal type."

The association of a streptococcal infection of the erysipelatous type is an almost inseparable and constant feature of elephantiasis.

As causes of non-filarial elephantiasis—the so-called *Elephantiasis nostras streptogenes*—"injuries involving the circumference of the limbs at their root, or tumours blocking the deep lymphatics; chronic ulcers of the leg, syphilitic, tuberculous and phagedenic in origin; chronic phlebitis associated with varices, post-typhoidal, or puerperal; chronic eczema and kindred conditions," are given.

Mention is then made of the different operations which have been employed in dealing with the condition. Those of KONDOLEON, LANZ, ROSANOW and others have already been dealt with [see this *Bulletin* Vol. I, pp. 93 and 421, 422], as have also the experiences of MADDEN, IBRAHIM and FERGUSON in Egypt with HANDLEY's lymphangioplasty operation [see this *Bulletin*, Vol. 1, pp. 92, 93]. As regards the results of his own case, in common with the opinion of the authors quoted, Matas believes that the evidence furnished is insufficient to prove that a curative method of permanent drainage in elephantiasis or in the elephantoid states has been found in KONDOLEON's operation or in the other procedures (LANZ, von OPPEL, ROSANOW) of which it is a derivative.

He points out "that the principle of lymphatic derivation, by removing the aponeurotic barrier which separates the stagnant from the normal circulating lymphatic areas, is not free from theoretical objection. The excised aponeurosis is bound to be replaced by cicatricial tissue, which may block the path of the lymph stream and venous circulation as completely as the normal aponeurosis. The possibility remains, however, that by the



massive removal of the obstructing aponeurotic barrier new anastomosing lymph channels and veins (which reproduce themselves with astonishing rapidity) may form between the vessels in the stagnant areas and those in the active, circulating, tissues, and that these will resist the contractile tendency of the newly formed cicatricial tissue. If in the meantime, all sources of dermal and hypodermal infection can be kept away by asepsis, and "antigenous" treatment, it is possible that a permanent drainage may be established. It is obvious that the possibilities of the new principle can only be tested by clinical experience and further observation," and it is with the hope of encouraging this that the author records these cases.

G. C. L.

**OUZILLEAU.** L'Eléphantiasis et les Filarioses dans le M'Bomou (Haut-Oubangui). Rôle de la *Filaria volvulus*.—*Ann. d'Hyg. & de Méd. Colon.* 1913. Vol. 16. No. 2. pp. 307-321.

The author deals fully with the question of elephantiasis and its treatment. The procedure adopted at Bangassou for the operation of elephantiasis of the scrotum and penis is given in detail, black and white sketches showing the lines of incisions and other points. In addition other forms of elephantiasis are described and pictured, lymph scrotum and adenolymphocele being included.

The disease as seen in the scrotum appears to be exceedingly prevalent at M'Bomou, BERNARD in 1911 having operated upon 154 cases (scrotum and penis 143, scrotum alone 5, penis alone 2, labium majus 4), while in 1912 the total number of operations for this condition was 182, the vast majority again being genital cases. The mortality after operation is insignificant. Elephantiasis is also seen in the limbs but is rare.

The paper is to be continued. No mention is made in this part of it of the supposed role of the *Onchocera volvulus* in the production of the disease [see this *Bulletin*, Vol. 1, p. 420].

G. C. L.

**KUHNE & GUHNE.** Zur operative Behandlung der Elephantiasis scroti. [Operative Treatment of Elephantiasis scroti].—*Arch. f. Schiff- u. Trop.-Hyg.* 1913. July. Vol. 17. No. 13. pp. 457-458.

The operative treatment of elephantiasis scroti is described. Some modifications of the usual technique are given.

G. C. L.

**LÉGER (André).** Microfilaires sanguicoles de quelques Oiseaux du Haut-Sénégal et Niger.—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 359-367.

The paper deals with different microfilariæ found in the blood of birds in the Upper Senegal and Niger, many of which were hitherto unknown. The author gives the scientific names of the birds and the characteristics of the different filariæ discovered in them, some of which have not hitherto been ascribed to these. [These filariæ are not mentioned in the subject should consult the original.]

## TROPICAL DISEASES BUREAU.

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## KALA AZAR.

## INDIA.

DONOVAN (C.). *Kala-Azar, its Distribution and the Probable Mode of Infection.*—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19 & 20, 1912.* pp. 211-214; and *Indian Jl. Med Research.* 1913. July. Vol. 1. No. 1. pp. 177-184. With a map.

In this paper the author reviews the distribution of kala azar both in and outside India. It appears uncertain whether the disease occurs in Orissa or not. He then goes on to consider the probable modes of infection under two headings: (1) By means of insects, (2) by oral ingestion.

He considers that the bed bug theory of transmission has yet to be proved and that the *Conorhinus rubrofasciatus*, suspected by him to be a transmitter, has not received such a fair trial of experiment as to justify its exclusion. Further, mosquitoes have been very little experimented with, in spite of the fact that kala azar and malaria exist side by side. As regards the flea and the possibility of its transmitting the disease from dog to man as it is supposed by some to do in Europe, the author points out that most extensive search has failed to reveal the canine disease in India even in the chief endemic centres of the disease. The author himself examined the spleen and liver of over 1,000 dogs in Madras in 1910, and Captain PATTON has recently examined a still larger number with negative results.

The infection of a pup four months old after intrahepatic injection of  $3\frac{1}{2}$  c.c. of spleen blood obtained from a case of kala azar is recorded. Two and half months after injection the dog was killed and parasites were found only in the bone marrow of the ribs while the liver and spleen were free. This fact renders it necessary to re-examine Madras dogs for the disease, since in previous examinations the bone marrow was only examined a few times. As regards the infection of the pup this is the first record of the experimental production of the disease in dogs. [PATTON also records the infection of dogs.]

As regards the possibility of oral infection the author notes that in a good percentage of cases of kala azar the illness commences with symptoms of intestinal involvement, and that in fatal cases ulceration of the gut is marked and leishmania occur in the tissues about the ulcers. It is suggested that possibly ankylostomes or even trichomonads might obtain parasites from the gut and transmit them.

C. M. Wenyon.

PATTON (W. S.). *Is Kala Azar in Madras of Animal Origin?—Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19 & 20, 1912.* pp. 215-220; and (in revised form) *Indian Jl. Med. Research.* 1913. July. Vol. 1. No. 1. pp. 185-195.

The author inoculated a series of animals (3 monkeys, 4 dogs, 2 jackals, 2 guinea pigs, 2 rabbits, 2 cats, 4 white rats, 1 goat, 1 pig, 1 calf) with comparatively large doses of emulsion of a very heavily infected spleen of a case of kala azar. The dose varied from 1 cc. in the case of the smaller animals to 4 cc. in the goat, 7 cc. in the pig and 9 cc. in the calf. All these animals were reinoculated fifteen days later with an emulsion made from another infected spleen. All three monkeys were infected, one dying after five weeks, another after six weeks, and the third killed six to seven weeks after the second inoculation. All the dogs were infected, the first dying 3½ months and the second four months after inoculation. In the first dog the liver, spleen, bone marrow and lymphatic glands contained many parasites while in the other dog there were *Piroplasma canis* as well as many leishmania. In films of the peripheral blood of this dog made a few hours before death both parasites were seen in polynuclear leucocytes. The author calls attention to the possibility of confusing phagocytosed piroplasma with leishmania. The third dog remained healthy for nine months and then became ill and died ten days later. Four days before death *Piroplasma canis* and a few leishmania were found in the peripheral blood; the next day 500 leishmania were found in a single blood film and 48 hours before death as many as 1,000 parasites were found in a single film. After death smears from the organs contained many parasites. Dog No. 4 was caged next the one just described and it contracted *Piroplasma canis* about the same time. It was killed nine months after the original inoculation and smears of the spleen, liver and bone marrow showed many leishmania. Two days before death a few leishmania were found in the peripheral blood.

One of the jackals became emaciated nine months after inoculation and a few leishmania, as well as *Piroplasma canis*, were found in the peripheral blood four days before death. After death many leishmania were found in smears of the organs. One of the white rats died two hours after receiving the second inoculation. Numerous leishmania were found in the liver and spleen (see this *Bulletin*, Vol. 1, p. 3). None of the other animals became infected nor a further series of monkeys, dogs and rats which were inoculated with smaller doses of the virus.

In addition to the above experiments the author has examined

the spleens of 1,438 dogs destroyed in the lethal chamber in Madras from January 29 to November 25, 1912, and the spleens and bone marrows of 1,321 dogs from November 26, 1912 to May 16, 1913, without finding a single case of infection with leishmania. During the past seven years about 300 cats have also been examined with negative results. From the above results the author feels justified in concluding that the dog and probably the cat play no part in the transmission of kala azar in Madras.

C. M. W.

PATTON (W. S.). **Further Observations on the Development of *Herpetomonas donovani* in *Cimex rotundatus* and *Cimex lectularius*.**—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19 & 20, 1912.* pp. 221-232.

The author refers to his previous work (see this *Bulletin*, Vol. 1, pp. 1-3), in which he has shown that the parasite of kala azar will complete its development in the bed bug into the post-flagellate form if the insect abstains from a second feed of blood during the course of this development. Having a case of kala azar, in the peripheral blood of which numerous parasites occurred, a series of bugs was fed with the object of determining the temperature most suitable for this development. After feeding, the bugs were divided into batches and each batch kept at a different temperature. Seven bugs were kept at a temperature varying from an average minimum of 85° F. to an average maximum of 94° F. In these the parasites flagellated but instead of multiplying died out in about five days. Some more bugs (number not stated) were fed on the case and kept at the same temperature as the others. Before dissection they were fed on a rat. When dissected nothing was found in them. Six bugs and some others [number not stated] were fed and then kept at a temperature varying from 75° F. to 80° F. In four of these dissected after 39, 42, 46 and 96 hours respectively flagellates were found. In one dissected after 96 hours a few degenerating parasites were seen and in two dissected after 96 and 149 hours no flagellates were found. Others dissected after 5, 6, or 7 days were negative. Though the parasites will flagellate at this temperature then they quickly die out. A series of 39 bugs were kept at a temperature of 73° F.-75° F. In these bugs also flagellation occurred, but the parasites appeared to die out after four or five days. Two bugs kept at a temperature of 55° F.-65° F. and dissected after 93 and 96 hours revealed only partly digested blood and parasites either free or in unaltered leucocytes. Another 24 bugs kept under the same conditions were dissected from the fifth to the tenth day. They contained unchanged parasites, developing forms and in each a few flagellates. The low temperature had evidently retarded development. Unchanged parasites were seen in some bugs kept at the low temperature after the twelfth and thirteenth days. Apparently none of the above temperatures are suitable for a complete development of the flagellate and

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prove to the author that the temperature limit for this development is a very narrow one. In his previous experiments (see this *Bulletin*, Vol. 1, p. 1) the author kept the bugs at a temperature of 71° F.-75° F. and in these a complete development to post flagellate forms occurred. This necessary range of temperature is to be found in Madras during the months of December and January and it is then that the parasite taken up from the peripheral blood by the bug is able to pass on through the flagellate to the post flagellate stage, provided that the bug does not have a second feed of blood before the post-flagellate stage is reached.

Apart from experimental work the paper contains some observations on the occurrence of parasites in the peripheral blood of kala azar cases. The case used for the experiments on one occasion showed as many as 1,043 parasites in a single blood film. This was four days before death. It was found post-mortem that in addition to a solid pneumonic lung a large empyema was present on the anterior surface of the upper lobe. In other cases the onset of severe dysenteric symptoms is accompanied by an increase in the number of parasites in the circulation. In one case in which this was found it was noted that the right kidney was practically a bag of pus. In all these cases there was a marked leucocytosis and this reaction caused the parasites to multiply in the spleen and possibly elsewhere. On being freed from the large endothelial cells they were taken up by the leucocytes and appeared in the peripheral blood.

C. M. W.

Cragg (F. W.). *An Investigation into Kala-azar.—Ibid.* pp. 39-42.

The fact that the development of any blood parasite in its transmitting host only takes place in a limited number of those actually taking up parasites from the blood led the author to make the following investigations. He studied the behaviour of the gut of flies after they had taken a full meal of blood, with a view of discovering in this behaviour an explanation of the non-development of the parasite in so many instances in which apparently circumstances were quite favourable for such development. For this investigation large Tabanid flies were used. The flies were given a full meal of blood and the guts, dissected out after varying intervals, were serially sectioned. The remarkable discovery was made that after the first feed of blood the whole of the epithelium lining the mid gut is thrown off into the lumen and as digestion proceeds a new epithelium is produced from nuclei which were previously lying between the columnar cells before desquamation had taken place. These changes do not occur in quite the same manner in flies which have a second or third feed of blood. This fact appears to the author to offer a possible explanation of the irregularity in development of any parasite in its transmitting host.

C. M. W.

MACKIE (F. P.). *Progress Report on Kala Azar.*—*Ibid.* pp. 233-238.

The report refers mainly to investigations on kala azar in Kalna, Burdwan district, where MUIR previously reported that he saw as many as six or seven hundred cases of kala azar in a week (*Indian Med. Gaz.* 1911. Feb., pp. 58-60 and *Kala Azar Bull.* 1912. No. 3. pp. 125-126). It was found that certainly four or five hundred cases of enlarged spleen passed through the dispensary each week. During two months 58 of these cases were suspected of kala azar and were subjected to spleen puncture with a positive result in 23 (39·6 per cent.). It was therefore evident that the great majority of unselected cases were not kala azar but probably malarial splenomegaly. An examination of 236 children in four schools in Kalna showed enlarged spleen in 23·2 per cent. None of the children could be suspected of having kala azar. Of the 23 cases diagnosed by spleen puncture none was under 10 years of age, 12 were between 10 and 20, 8 between 20 and 30, and 3 over 30. In pursuing such investigations in a country riddled with severe malaria the only safe criterion of kala azar is the discovery of leishmania, for the author has been deceived by clinical symptoms on several occasions. There could be found no special focus of the disease, which seemed to be scattered irregularly throughout the district without apparent reference to any climatic or geographical conditions. There was further no particular tendency to house infection. In many cases four or five apparently healthy persons were living in the same house with a proved kala azar patient. In Madras and Assam on the other hand house infection is said to be common.

As regards clinical symptoms and signs the author was much struck by the absence in the Kalna cases of some of the most marked features of the disease as ordinarily described. Amongst the 23 cases seen only three could be described as classical kala azar. In a good number of the Kalna cases the patients were fat and robust, bright and cheerful, and beyond enlarged spleens and irregular fever showed no signs of disease. Some of such cases showed large numbers of leishmania on spleen puncture. Recoveries are said to be frequent. It is possible that the disease is milder in Bengal than in Madras or that only advanced cases came into the Madras Hospital.

*Examination of the peripheral blood* has not been used as a diagnostic method as spleen puncture was available. It was carried out however in most of the severe and advanced cases, but parasites were found in two cases only and these in small numbers. The author had no difficulty in finding parasites in the peripheral blood of Madras cases, so does not consider the failure in Kalna as due to technique but rather to the greater chronicity and mildness of the disease in Bengal.

*Experiments to ascertain whether bed bugs harbour the parasite of kala azar.*—321 bugs taken from the beds and bedding of patients proved to be suffering from kala azar were dissected with negative result, while 70 laboratory-bred bugs were fed on



a patient in whose peripheral blood leishmania had been discovered (5-6 per slide). They were allowed to feed once and kept at ordinary air temperature. They were dissected from 1-30 days after feeding with the result that leishmania of the usual form were found in one or two on the day after feeding but not subsequently. Similar experiments with seven leeches were made with results so far negative. An examination of 22 bazaar dogs gave negative results as regards leishmania.

*Animal inoculations* were undertaken with the spleen juice of a boy who had died of kala azar. Thirteen white mice were inoculated. Only one became infected after intraperitoneal injection. It wasted and died seven days later and leishmania were found in smears of the spleen only. Two flying foxes were injected intraperitoneally. One of these died 16 days after inoculation, when an abscess was found in the abdominal wall. In the abscess pus some typical leishmania were found. The second flying fox was killed three weeks after inoculation. The only organs in which leishmania could be found were the pancreas and kidney. One large monkey received 10 cc. of spleen emulsion by the mouth. Two months later the animal was killed but no leishmania were discovered. Two other monkeys were injected intraperitoneally with 5 cc. of emulsion. One was killed two months and ten days later with no result, while the other was found by liver puncture to harbour leishmania one month after inoculation. Two months after inoculation it still appeared quite healthy though the spleen was enlarged and palpable. Puncture of this organ revealed leishmania. Ten days later it was killed and fair numbers of parasites were found in the spleen, few in the liver and none in the bone marrow or any other organ. Seven white rats were inoculated with a negative result in each case.

C. M. W.

KORKE (Vishnu T.). Progress Report on "Some Observations on the Epidemiology of Kala Azar in Madras."—*Ibid.* pp. 239-256.

In Madras that part of the town known as Georgetown is the cradle of kala azar and during the last nine years the disease has not diffused evenly more than two miles in any direction from this centre. Cases from the outskirts of the city are exported from the centre, though Triplicane is an exception in that a small endemic centre exists here. The average number of persons living in each house in Georgetown is thirteen and this overcrowding together with defective hygiene is a very important fact in relation to the disease. In this district kala azar is purely *endemic* without any tendency to assume an *epidemic* form. Outside the city of Madras kala azar occurs in the Presidency, for the author saw two cases, one from Cuddalore, the other from Madura, and met with another at Trichinopoly—a ~~fact~~ from Malabar: suspected cases were also seen at Mandapan. As regards seasonal incidence of the disease nothing definite could be determined, as the generally accepted view that all nationalities of all sexes and ages are affected was confirmed.

A detailed study of kala azar in its relation to the houses was undertaken, since it has been believed that houses themselves play an important part in the spread of the infection.

The result of this enquiry has been to establish the fact that unless case to case or contact infection has taken place further cases have not occurred in the houses. Thus, no case of the disease was found among the fresh tenants of 26 houses in which more than one case had occurred and in 300 houses in which only one case had occurred and of which the present inhabitants have never come in contact with a kala azar case.

In an examination of the cases with a view to determining the incubation period of the disease it was found that in some cases there was a minimum period of four months' incubation while in others it was much longer. From clinical and other observations it would appear, as DONOVAN and PATTON have already pointed out, that there is a particular period in the course of the disease which is more infective than the rest.

C. M. W.

YOUNG (T. C. McCombie). **An Account of an Investigation of the Prevalence of Endemic Kala Azar in the Plains of Assam.**—*Ibid.* pp. 257-265.

The epidemic of the 'eighties and 'nineties commenced in Goalpara and spread up the Brahmaputra Valley *via* the grand trunk road on the south bank of the river, through the sub-divisions of Dhubri and Goalpara to Kamrup. It then crossed to the north bank into Mangaldai and thence passed on to Nowgong in 1890. By 1896 the disease had reached the narrow tract of sparsely populated country which gives access to the more open, alluvial plains of the Golaghat sub-division and the Upper Brahmaputra Valley. Here stringent measures stayed the epidemic and the Upper Assam Valley was saved from its ravages. Subsequently the disease in its epidemic form declined and in 1901 it was considered that Goalpara and Kamrup were free from epidemic. Endemic foci however have been left behind and the present investigation indicates the position of these.

In the Golaghat sub-division of Sibsagar where the epidemic finally burnt itself out endemic centres still exist. This sub-division consists of 21 "mauzas" containing 629 villages, of which four hill mauzas were excluded from the survey and 396 villages were examined. In the Khumtai "mauzas" two villages have 23 infected houses while in the Maharani mauza two villages have nineteen infected houses. Four other villages also are infected. There is now accurate knowledge of the conditions in Golaghat and this is kept up to date by a Sub-assistant Surgeon. There is no cause for anxiety as to the present condition of affairs although the situation requires vigilance.

In Kamrup kala azar is prevalent in a village in the "thana" of Rangiya, in Sylhet a considerable number of deaths from the disease has been reported (1910), in Goalpara the disease was reported in the Dudnai thana (1911-12) and in Mangaldai the disease has recently been noted.

The author writes that the present situation appears to be this:—"that in an unknown number of areas in the Lower Brahmaputra Valley and throughout the Surma Valley there are still glowing points of slow combustion endemic foci. Furthermore in the Upper Assam Valley, which is the main centre of the important tea industry, there exists a population so far untouched by the endemic form of the disease, and hence presumably a rapidly combustible material for an epidemic fire, and that adjacent to this inflammable material there exists in Golaghat an area of combustion of not inconsiderable activity." At present the survey is continuing with a view to mapping out accurately all endemic foci so that the question of controlling and dealing with them in the future will be enormously simplified.

C. M. W.

BOSE (Kailas Chandra, Rai Bahadur). *The Relation of Kala Azar to Malaria.*—*Ibid.* pp. 267-270.

It is often very difficult to separate the two diseases kala azar and malaria. A case which starts clinically as one of malaria with fever of the intermittent or tertian type may change its character and become one of kala azar with quotidian or continued fever. Apparently the author thinks it possible that at the time the fever takes the continued course the organisms of one class become replaced by organisms of the other so that quinine ceases to be of any avail. The author favours the treatment of these cases with measures calculated to produce a leucocytosis, for in his experience recoveries are by no means uncommon.

C. M. W.

MUIR (E.). *The Diagnosis and Treatment of Chronic Malaria and Kala-Azar.* [*Mirror.*—*Indian Med. Gaz.* 1913. July. Vol. 48. No. 7. pp. 267-268.]

The author refers to his previous paper on the subject of kala azar in Kalna, Burdwan District (see *Kala Azar Bulletin*, No. 3, pp. 125-126), in which he advocated the treatment of the disease by the intramuscular injection of acid solutions of quinine with a view to producing a leucocytosis. Formerly, the author did not regularly diagnose the cases by the discovery of the parasite, but since the visit of Captain MACKIE (see Report by MACKIE, reviewed in this number) he has performed spleen puncture 150 times and is convinced that there is not the least danger attending this operation, at any rate in the cases in the Burdwan and Hooghly districts of Bengal in which the spleens are firmer than they appear to be in the Madras cases. A small needle must be used and only a drop or two of pulp and blood drawn up. In a few very acute and advanced cases the spleen is soft, but in these the parasites can usually be found in the peripheral blood. The injections of quinine described in the earlier paper continued to be of great benefit in cases of kala azar which are seen in time. The chief objection is that the injection is very painful for a few minutes if local anaesthetics are not used and this

is a difficulty in dispensary practice, in which one injects more than 100 patients in a day. Lately the author has injected another mixture consisting of:

Turpentine	...	...	...	...	1 drachm.
Camphor	...	...	...	...	1 "
Creosote	...	...	...	...	1 "
Sterilised olive oil	...	...	...	...	2½ "

The camphor and creosote are mixed first and the turpentine and oil added; 5-15 minims are injected into the muscles on both sides of the body, the latissimus dorsi or gluteus being most suitable. There is very little pain at the time, but later a certain amount of pain and swelling appears. Apparently the author uses this treatment in chronic malaria as well as kala azar, for he says that the reaction is much more marked in chronic malaria than kala azar in which larger doses have to be used to produce the same effect. In kala azar it is necessary to repeat the injections more frequently and to persist in them for a much longer time. In advanced kala azar these injections fail to cure though they may produce some temporary improvement. The onset of kala azar is usually with symptoms very like typhoid, with the exception that the tongue is not so coated and there is an absence of Widal's reaction. If these cases can be diagnosed at the onset or even in the first relapse the author believes that the majority can be cured by the above recommended treatment.

C. M. W.

#### MEDITERRANEAN.

**SOCIETÀ ITALIANA FRA I CULTORI DELLE MALATTIE ESOTICHE.**  
**Riunione Privata tenuta a Messina il 15 Giugno 1913 intorno alla Leishmaniosi Umana in Italia.** Atti, Relazioni, Comunicazioni Scientifiche (con tavole) per cura del Prof. Dr. G. Spagnolio, Dr. M. Signer, Segretari della Riunione.—180 pp. With 5 plates and 4 maps. 1913. Messina: Stab. Tipografico Guerriera. [Kala Azar. pp. 17-152.]

i. SCORDO (F.). **L'Etiologia della Leishmaniosi interna nel Bacino Mediterraneo.** pp. 17-49.

This paper is a very interesting summary of the present state of our knowledge of Mediterranean kala azar.

ii. DI CRISTINA. **Sulla Sindrome, Diagnosi, Prognosi e Cura dell' Anemia da Leishmania (Leishmaniosi interna).** pp. 50-63.

Like the preceding paper this is a review of results already published.

iii. GABBI (U.), PELLEGRINO (P. L.), & MONTORO (G.). **Inchiesta intorno al Kala-azar nelle Provincie della Sicilia Orientale e della Calabria Inf.** pp. 63-77: also *Malaria e Malat. d. Paesi Caldi*. 1913. June-July. Vol. 4. No. 4. pp. 239-253.

The authors discuss in detail the distribution and incidence of kala azar in Sicily and Italy. As far as can be determined

from statistics it is shown that about 60-70 children die of the disease each year in Messina and the surrounding villages with a population of about 128,000. In Catania about 100 are infected each year while in Palermo 92 cases have been encountered in three years, so that each year in the three cities of Messina, Catania and Palermo with their villages about 200 children die of kala azar. There remain in Sicily the towns of Syracuse, Trapani, Girgenti and Caltanissetta, which have not been thoroughly investigated though isolated cases have been encountered in the last three. As regards the neighbouring coast of Italy there is still greater uncertainty. La CAVA finds four or five cases each year in Bovalino with a population of 5,000 so that he estimates that in the whole of the Reggio Calabria district with a population of 200,000 the yearly incidence of the disease may be placed at 100. Extending these results to the coast of Tirrena and Jonica di Catanzaro with Lecce and the surrounding coast and adding the cases at Naples, Salerno and Caserta it comes about that in the provinces of Messina, Catania, Palermo, Girgenti, Trapani, Caltanissetta, Reggio Calabria, C'antauzaro, Lecce, Napoli, Caserta and Rome the number of cases each year must be put down at about one thousand.

- iv. DIONISI (A.). *Contributo alla Anatomia Patologica dell'Anemia da Leishmania*. pp. 78-81: also *Malaria e Malat. d. Paesi Caldi*. 1913. June-July. Vol. 4. No. 4. pp. 265-269.

The author describes the changes in the various organs and the distribution of the leishmania in the body. The changes are attributed partly to the anemia and partly to the parasitic infection. The parasites may be found in any organ in the endothelial cells of the blood capillaries, in the cells of the stroma of the lymphatic and blood forming organs, in the cells of the spleen pulp and sometimes in the renal epithelium and more especially in that of the loops of Henle. The author concludes that the histological changes are very variable so that diagnosis from these alone may be exceedingly difficult.

- v. CANNATA (S.). *Il Sangue nell'Anemia da Leishmania*. pp. 82-88.

This paper is a resumé of work previously published by the author and others, and has been already noted in the *Kala Azar Bulletin* and this *Bulletin*.

- vi. CANNATA (S.). *Sul Rapporto del Parassita di Leishman nel Sangue Periferico*. pp. 89-92: also *Malaria e Malat. d. Paesi Caldi*. 1913. Aug.-Sept. Vol. 4. No. 5. pp. 303-306.

This paper together with a previous note in *Pathologica*, 1913, June 15, and a paper in *La Riforma Medica*, 1913, August 2, deals with the demonstration of leishmania in the peripheral blood of cases of Mediterranean kala azar. Readers are reminded that in India and the Sudan parasites have been found in the peripheral blood of cases of the disease while in the Mediterranean districts NICOLLE alone has succeeded in demonstrating the parasite in the peripheral blood of two of his cases. The author

of the papers now under review has been successful in finding parasites in the peripheral blood in seven out of eight cases of infantile kala azar. The ages of the children examined varied from one to three years and the duration of the illness from twenty days to one year so that apparently parasites are present in the circulation at all stages of the disease.

Various methods of centrifugation were tried without success as it was found impossible to obtain satisfactory films showing clear and unaltered leucocytes. Accordingly ordinary thin quickly dried blood films were used and 20 to 30 were prepared from each case. A mechanical stage was used for the search which the author admits to be very tedious and to require much patience. In one case one slide showed a large mononuclear cell and a polynuclear with one parasite each, while another slide contained a large mononuclear with one parasite. In all the other cases only a single cell, either a mononuclear or polynuclear, containing one or two parasites was found.

vii. CARONIA (G.). *L'Anafilassi nella Leishmaniosi Infantile.* pp. 92-98: also *Pathologica.* 1913. July 15. Vol. 5. No. 113. pp. 420-423.

The fact that Di CRISTINA has expressed the view that the fever in infantile kala azar was due to anaphylactic phenomena led the author to investigate the anaphylactic properties of the blood in cases of this disease. To this end two methods of investigation were followed. In the first attempts were made to immunise guinea-pigs by injection of living cultures of leishmania till the animals ceased to react thermally to these injections. When in this condition 1-2 cc. of blood serum from a case of kala azar was injected into the animal intravenously. The animals reacted only by showing a slight fall of temperature and mild tremors of short duration. Accordingly the second method was adopted. This consisted of the intravenous injection of guinea-pigs with mixtures of 2-3 cc. of blood serum of a case of kala azar and 1-2 cc. of culture of leishmania on NNN medium. By this method much more definite results were obtained. Eight cases of kala azar were tested and in every instance the animals reacted definitely by showing tremors, dyspnoea, paralysis of the sphincters and a fall of temperature. This was followed by collapse and in those cases in which the largest dose was used (3 cc. serum with 2 cc. culture) death resulted. Four control experiments were undertaken wherein either healthy children or those suffering from other diseases were employed. In none of these was any such striking result obtained so that the author concludes:—

1. That the blood serum of children suffering from kala azar contains special substances capable of producing *in vitro* anaphylactic phenomena.

2. That all the symptoms shown by children suffering from this disease, especially the fever and the cachexia, are referable to such phenomena.

3. That this reaction may be used as a means of diagnosis when spleen puncture has been negative, as is the case sometimes in the last stages of the disease.

- viii. CARONIA (G.). *Sul Potere Complementare del Siero di Sangue nella Leishmaniosi Infantile.* pp. 98-102: also *Malaria e Malat. d. Paesi Caldi.* 1913. Aug.-Sept. Vol. 4. No. 5. pp. 309-313; and *Pediatria.* 1913. Aug. 31. Vol. 21. No. 8. pp. 583-587.

The investigations here recorded were conducted in the following manner. With all aseptic precautions a sufficient quantity of blood was drawn off from the finger of children suffering from kala azar. Healthy children were used as controls. The serum was allowed to separate and was then distributed in each case in a series of tubes, the first tube of the series receiving 0.01 cc. serum and the last 0.15 cc. The liquid in each tube was then made up to 1 cc. by the addition of physiological saline. To each tube was then added 1 cc. of a 5 per cent. solution of washed ox blood corpuscles together with 0.1 cc. of antibovine haemolysin of strength 1 : 1000. The contents of the tubes were then mixed and incubated at 37° C. for one hour. They were then examined and of those tubes showing complete haemolysis the one was selected to which the least quantity of original serum had been added. This quantity was taken to represent the complement power of the blood of the case of kala azar. In all sixteen cases were thus examined. The ages varied from eleven months to three years and eight months and the duration of the disease from twenty days to sixteen months. The highest value obtained was 0.07 and the lowest 0.03 with an average of approximately 0.04. In seven controls in healthy children the values were much higher, averaging 0.12. The author concludes that the complement power of the blood is increased in cases of kala azar and is more marked in the later stages of the disease.

- ix. SPAGNOLIO (G.). *Sulla Ganglio-puntura nella Diagnosi di Leishmaniosi.* pp. 102-104: also *Malaria e Malat. d. Paesi Caldi.* 1913. Aug.-Sept. Vol. 4. No. 5. pp. 306-308.

Many observers have noted that the lymphatic glands are enlarged in cases of infantile kala azar and COCHRAN in China has advocated the excision of the superficial cervical glands as a means of diagnosis in this disease. The author has examined two cases by puncture of lymphatic glands. In one of these a cervical gland and in the other an inguinal gland was punctured with the result that numerous leishmania were discovered in each case.

- x. SCORDO (F.). *Alterazioni Morfologiche dei Corpi del Leishman nel Kala Azar.* pp. 105-108: also *Malaria e Malat. d. Paesi Caldi.* 1913. Aug.-Sept. Vol. 4. No. 5. pp. 313-317.

The author notes that the parasites seen in smears of spleen material taken from certain severe cases of infantile kala azar have a peculiar degenerated appearance and that in some smears the non-nucleated fragments of leucocyte cytoplasm containing parasites are very numerous. He was led to think that should the degenerative process become more marked there might be a total disappearance of the leishmania, as apparently occurred in

a case recorded by LIGNOS (see this *Bulletin*, Vol. 1, p. 4) in which no parasites could be found at the autopsy through they had been shown by spleen puncture to be exceedingly numerous before death. According to the author this degeneration of the leishmania may take place in three ways. Firstly there is hypertrophy of the parasite followed by vacuolation of its cytoplasm; the nuclei increase in size, the kinetonucleus fragments, and finally after breaking up of the trophonucleus into small granules the parasite disintegrates. In the second method of degeneration the parasite becomes elongated and assumes the form of the flagellates seen in cultures on NNN medium though the flagellum is lacking; these become vacuolated and break up as before. Finally some of the parasites show a reduction of the cytoplasm, which eventually is reduced to a mere membrane enclosing the two nuclei. The author does not speculate as to the ultimate fate of these atrophic forms. The various degenerative processes are illustrated in a coloured plate. Figures are also given showing the detachment of portions of cytoplasm from large mononuclear cells containing parasites, giving rise to the bodies which have been described by various observers. The parasites may exhibit the degenerative changes while still enclosed in these portions of cytoplasm.

The author does not think that these degenerative phenomena are of rare occurrence and he is led to wonder if a periodic degeneration of parasites occurs during the course of the disease. He admits that these points can only be settled by further research.

**xi. RANIERI (G.). Il Primo Esempio di Leishmaniosi Interna a Campo Calabro. pp. 108-109.**

A case of kala azar in a child born in September, 1909, and the first to be recorded from Campo Calabro in Southern Italy.

**xii. LICCIARDI (S.). Sulla Leishmaniosi Interna in Catania. pp. 110-115.**

This paper is concerned with a case of recovery—the first to be recorded—of a child suffering from infantile kala azar, an account of which the author published in April, 1910. Since that time he has had a second case of recovery out of a total of 26 cases seen by him in Catania. He refers to cases recorded by JEMMA, NICOLLE, SPAGNOLIO, LIGNOS, CARYOPHYLLIS, SOTIRIADES, and PETRONE, bringing the total recorded cases of recovery to sixteen. [Some further cases of recovery are noted in the present number.]

**xiii. TIMPANO (P.). Un Caso di Leishmaniosi Interna finito con la Guarigione. pp. 115-116.**

The case recorded is one of an adolescent thirteen years of age, who suffered from irregular fever with large spleen, emaciation and marked anaemia. The case was thought to be one of malaria



but was treated without result with quinine. Spleen puncture was accordingly performed and leishmania discovered. The illness lasted three years and the case ultimately recovered, but the author is unable to say whether this was due to the treatment with quinine, arsenic and iron which had been carried out.

xiv. SPAGNOLIO (G.). **Nuovi Casi Clinici a Nicotera Marino ed a Nizza Sicilia.** p. 117.

Two cases of kala azar are recorded—one from Nicotera Marino in Calabria and the other from Nizza in Sicily. They are the first instances of the disease noted from these towns.

xv. VADALÀ (P.). **Il Primo Esempio di Kala Azar a Furnari dimostrato colla Spleno-puntura.** pp. 119-121.

Two cases of infantile kala azar, the first to be recorded from Furnari in Sicily, one of which was diagnosed by spleen puncture.

xvi. SPAGNOLIO (G.). **La Cura Climatica nella Leishmaniosi interna.** pp. 121-123.

The main object of this paper is to record the result of the treatment of eight cases of infantile kala azar by removing them to healthy surroundings in a sanatorium about 900 metres above sea level. The treatment did not give encouraging results as there seemed a tendency for broncho-pneumonia and intestinal derangements to occur more frequently. Two other cases from Bordonaro, after treatment with atoxyl, appear to be on the way to recovery.

xvii. ABATE (A.). **Contributo alla Casistica e allo Studio della Leishmaniosi Infantile.** pp. 123-126.

This paper is mainly a clinical study of 44 cases of infantile kala azar observed by the author in Catania. One interesting observation, which the author rightly considers to be unique, is the occurrence in one house of four children suffering from the disease. Diagnosis was made by spleen puncture in two of these only, but the author states that in the others the typical clinical picture could leave no room for doubt. Of the 44 cases only one can be regarded as having recovered.

xviii. ABATE (A.). **La Resistenza dei Globuli Rossi nella Leishmaniosi Infantile.** pp. 126-127: also *Malaria e Malat. d. Paesi Caldi*. 1913. June-July. Vol. 4. No. 4. p. 263.

This is a preliminary note dealing with investigations into the resistance of the red blood corpuscles in twenty cases of infantile kala azar. The method employed was CUTORE's modification of that of VIOLA. The results are not in accord with those of CANNATA, for the author finds that the resistance is increased, especially as regards the maximum and minimum resistance.

- xix. LA CAVA (F.). **Un Caso di Leishmaniosi Interna (Kala-azar) in una Giovinetta di 14 anni.** pp. 127-130: also *Malaria e Malat. d. Paesi Caldi*. 1913. Aug.-Sept. Vol. 4. No. 5. pp. 317-320.

A case of kala azar in a female child aged 14 years is described in detail.

- xx. PUGLIATTI (G.). **Sulla Ricorrenza Primaveraile della Leishmaniosi Interna.** pp. 130-133.

Four cases of infantile kala azar seen by the author appear to support the view that the disease commences between the months of February and June with a maximum number of cases in April and May.

- xxi. SIGNER (M.). **Sulla Distribuzione della Leishmaniosi in Italia.** pp. 133-135: also *Malaria e Malat. d. Paesi Caldi*. 1913. Aug.-Sept. Vol. 4. No. 5. pp. 320-323.

The author considers the distribution of kala azar in Italy and Sicily and illustrates his remarks by two maps. He points out that the disease both in Italy and Sicily is found almost exclusively near the sea. This is true also of the whole Mediterranean district and suggests to the author that the disease is carried about by shipping and that with increased facilities of communication it will be likely to spread to inland towns.

- xxii. GABBI (U.). **Sulla Identità Clinica ed Etiologica della Leishmaniosi mana e Canina.** pp. 136-147. With 7 tables: also *Pathologica*. 1913. Sept. 15. Vol. 5. No. 117. pp. 543-552.

The paper is a lengthy study of the published experiments and observations dealing with the relations of human to canine kala azar. The author shows that according to results so far obtained there are marked differences between the spontaneous or naturally acquired kala azar of dogs and that artificially produced by inoculation of virus from cases of the human disease. In a table are set forth the differences in the symptoms of the disease in man and dogs; it is shown that many of the symptoms found in man are wanting in dogs and *vice versa*. Another section of the paper deals with the inoculations which have been done on different animals with the human virus, the cultural forms of this, the canine virus and its cultural forms. A series of very interesting tables is given showing the results of these various inoculation experiments, the animals used and the author making the experiments. As a result of the enquiry the author finds that there is no agreement between the results obtained with the different viruses as one would expect to be the case if they were identical. It is concluded that in the present state of our knowledge neither the symptoms nor the pathology lend colour to the view that human and canine kala azar are one, and that too many gaps exist in the experimental work to justify any definite assertions

and much less the establishment of any basis of prophylaxis. The various tables compiled, evidently with much trouble, from previous publications are of much interest to those engaged upon similar work but they are too long to be reproduced here.

xxiii. SPAGNOLIO (G.). *Leishmaniosi Umana e Canina.*—Studio d'Ambiente. pp. 147-152.

In this paper the author contests the view that infantile kala azar is of canine origin and that fleas are responsible for the transmission of the malady. In Bordonaro, near Messina, during 1912 three cases of infantile kala azar were discovered and only in one case was a dog kept in the same house and it was quite healthy. Further, during last January, February, and April, BASILE has discovered in this village five dogs infected though no case could be found amongst the children who had associated with these dogs. The author thinks that if infection comes from the dogs five heavily infected dogs should be sufficient to spread the disease broadcast amongst other dogs and children. This apparently has not been the case.

It has been claimed by many observers that infantile kala azar commences in the early months of the year, in spring, and BASILE has stated that the statistics relating to the occurrence of canine kala azar indicate that dogs also contract the infection during this period, since the greatest number of dogs infected have been found in the months of June and July. To the author it seems clear that children become infected in January, February and March since most cases appear in March, April and May and this latter period corresponds to the period of incubation in dogs, so that it can hardly be assumed that children contract the disease from dogs. Further these periods should correspond to the greatest period of activity of fleas, which they do not, for fleas are most active in summer. It is pointed out that one cannot obtain good evidence of transmission of infection from one child to another, that the cases in adults are not in women who naturally harbour more fleas than men and who have been nursing infected children, that the occurrence of several cases at one time in the same family is not the rule, that there is no regular association of infected dogs with infected children, that if fleas were the transmitting agents the disease should be more wide-spread than it is, that in any locality the cases occurring in succession are not near one another but are far apart,—these are the chief arguments brought forward in this paper against the canine origin of infantile kala azar. [Perhaps the writer may state that in his opinion the knowledge of many of these subjects is as yet too incomplete to admit of conclusions being drawn, especially in reference to the periods of incubation in man and dogs, the susceptibility of dogs and children to infection and the percentage of the transmitting hosts (whatever this may be) which become infective even after taking up parasites. The disease in children and dogs may still be the same disease without it being necessary to prove that children must necessarily contract the disease from dogs. An infected dog would be a danger not only to children but to other

dogs as well, in just the same way as an infected child would be a danger to any susceptible dog or child with which it might come in contact].

C. M. W.

PITTALUGA (G.). *El Kala Azar Infantil (Esplenomegalia Parasitaria de los Niños) en la Costa de Levante de España.*—*Revista Clínica de Madrid.* 1912. Oct. 1. [7 pp.]

PITTALUGA (G.), del DIESTRO (J. García), & VIIÁ (Manuel). *Estudios sobre el "Kala-azar infantil" y la "Leishmania infantum" en España.*—*Boletín del Instituto Nacional de Higiene de Alfonso XIII.* 1912. Dec. No. 32. pp. 17-45. With 3 plates.

These two papers have to do with the discovery of infantile kala azar in Spain. In the first of these it is shown that a disease which is undoubtedly kala azar has been long recognised on the Mediterranean coast of Spain. Thus in Perello, which has a population of about 2,000, since the year 1904 42 cases have been recorded under various names in children varying in age from a few months to three years, which, in the light of more recent research, should be regarded as infantile kala azar. In one case from Tortosa leishmania were discovered by spleen puncture, thus demonstrating that an endemic centre of the disease exists on the Levant coast of Spain.

In the second paper four further cases of this disease are described. The clinical manifestations are the same as those observed in cases from other parts of the Mediterranean and in three of the cases leishmania were discovered by spleen puncture. Cultures of the organism were obtained on NNN medium and these have been maintained through several sub-cultures. In connection with the preparation of this medium the authors point out that most of the rabbits employed are infected with *Trypanosoma cuniculi* and that its presence might lead to confusion with the cultural forms of the leishmania. In practice however the temperature of preparation of the medium usually kills off the trypanosomes, but where this does not occur it is easy to distinguish the trypanosomes from the leishmania.

Eight dogs from houses in which cases of splenomegaly occurred were examined with negative results. One hundred mosquitoes (*Culex* and *Anopheles*) were taken from a house in which a case of kala azar occurred and in one of these parasites resembling leishmania were found.

The bulk of the paper is occupied with a description of the cases, three of which are shown in photographs, and the leishmania obtained from them. Apart from the discovery of an endemic centre on the East Coast of Spain the paper contains little new information.

C. M. W.

**BASILE (Carlo).** I recenti Studi sull'Identità della Leishmaniosi Umana e Canina del Mediterraneo.—*Policlinico*. Sez. pratica. 1913. July 20. Vol. 20. No. 29. pp. 1029-1032.

This paper is a review of recorded observations upon kala azar in the Mediterranean districts with the object of proving that the disease is the same whether in children or dogs. No new arguments in favour of identity are produced, but the author goes over the old ground and discusses the disease from the point of view of distribution, seasonal recurrence, symptomatology, therapeutics, parasitology and transmission. The author is convinced of the identity of the human and canine diseases.

In connection with the village of Bordonaro near Messina with a population of 2,000 the number of infantile cases found in the four years 1909-1912 has been 7, 1, 3, 3, and for the five years 1909-1913 the cases in dogs have been, 27 positive out of 33 examined, 4 out of 4, 5 out of 6, 7 out of 7 and 3 out of 3; so that from these figures the canine disease is still very prevalent there. It is pointed out that these examinations were made on dogs with clinical signs of the disease so cannot be taken as percentages of all dogs in the village.

C. M. W.

**TOMASELLI (A.).** Le Complicazioni della Leishmaniosi infantile.—*Malaria e Malat. d. Paesi Caldi*. 1913. Apr.-May. Vol. 4. No. 3. pp. 180-181.

This paper gives a list of the various complications which have been noted by different Italian writers in kala azar in Italy. Apparently peritonitis resulting from perforation of the gut is the only complication of Indian kala azar which has not been noted in the Italian form of the disease. The paper contains no original matter.

C. M. W.

**QUILICHINI.** Un Cas de Leishmaniose infantile suivi de Guérison. Formules Leucocytaires dans la Leishmaniose.—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 495-498. With 1 curve.

The present account refers to a case of infantile kala azar from Algiers, which was the subject of a former paper by the author in conjunction with the brothers SERGENT and LOMBARD and was of especial interest in that a dog and cat living on the same premises were found to be infected also (see *Kala Azar Bulletin*. 1912. No. 3. p. 133). The author notes that during the early part of February 1912 the child's condition was so bad that they considered it had reached the final stage of its illness and that recovery was impossible. In spite of this active treatment was adopted with a most pleasing result. The child's weight being 8.5 kilos, an intramuscular injection of 0.08 cg. of hectine was given on the first day and repeated daily, the dose being increased each time by half a centigram till 0.10 cg. was reached. This dose was given for ten days. The fever was not appreciably affected by the treatment though the general glandular enlargement was

diminished and the spleen slightly reduced. In spite of a complete tolerance of the drug it was thought advisable to discontinue the injections of hectine for a few days. Accordingly daily injections of 1 cg. in 1 cubic centimeter of "*iodone injectable Robin*" were made. This had hardly any influence on the temperature, but the glands of the groin which were enlarged became completely reduced, while the spleen rapidly decreased till it was not more than three fingers' breadth below the costal margin though previously it had been enormously enlarged, extending to the iliac crest. After ten injections of iodine the hectine treatment was repeated for ten days. The spleen continued to diminish rapidly. This treatment of alternating injections of hectine and iodine was continued for three months, after which the fever disappeared completely, the spleen and liver assumed their normal proportions and the general health of the child improved rapidly, while its weight was increased by 2 kilos in one month. Though recovery now seemed assured, injections of hectine and iodine were continued for four months longer with increasing intervals between the injections. Fifteen months have now elapsed since the child ceased to have any clinical signs of the disease. The author thinks that the treatment adopted contributed largely to this remarkable recovery.

C. M. W.

**LIGNOS (Antoine).** *Deuxième Cas de Guérison de Kala Azar infantile observé à Hydra.*—*Bull. Soc. Path. Exot.* 1913. June. Vol. 6. No. 6. pp. 430-432. With 1 chart.

During the year 1910 six children suffering from kala azar came under the author's notice in Hydra. Of these five have died and one has recovered. During 1911 thirteen cases were encountered and of these eleven have died, one is alive (apparently uncured) and one has recovered. The first case of recovery was recorded in a former paper (see *Kala Azar Bulletin*. 1912. No. 3. pp. 132-133). A detailed account of the second case is now given. The child was eleven months old and was apparently in perfect health when it became suddenly ill on December 15th 1911. Leishmania were discovered by spleen puncture on January 28th, 1912. The child's condition fluctuated during the next six months and in August it was taken by its parents into the country. It was brought back in October free from fever and in a very satisfactory condition. Since that date all symptoms have disappeared and the spleen is no longer palpable. The recovery is attributed to the fact that the child was kept in the open air from August onwards.

C. M. W.

**CARONIA (G.).** *Curve Termiche nella Leishmaniosi infantile.* [Temperature Curve in Infantile Leishmaniasis.]—*Pediatrics*. 1913. July 31. Vol. 21. No. 7. (2a Serie, Vol. 11.) pp. 481-496. With 12 curves.

A detailed study of twelve cases of infantile kala azar is given with a view to discovering any particular features of the temperature curves in this disease. It is impossible to review this

paper here. The author's conclusion is that the temperature-curve, from whatever point of view it is studied, has such special characters and is so distinct from all types of fever known that a careful and prolonged observation of it would place one in the way of a correct diagnosis before spleen puncture was adopted.

C. M. W.

CANNATA (S.). *Inclusioni Leucocitarie nella Leishmaniosi Infantile.—Pathologica.* 1913. July 15. Vol. 5. No. 113. p. 420.

The author has noted that in cases of infantile kala azar the polynuclear leucocytes frequently contain granules of various shapes in their cytoplasm. They are either oval, coccoid, rod-shaped or in the form of slightly curved filaments and are either nuclear fragments phagocytied by the leucocytes or they may actually be remains of ingested leishmania. Similar inclusions have previously been described by DÖHLE in scarlatina and by others in a variety of pathological conditions.

C. M. W.

LAVERAN (A.) & NICOLLE (C.). *Le Kala Azar Méditerranéen ou Infantile.—Trans. xvii Intern. Congress of Med. London.* 1913. Section xxi. Trop. Med. & Hygiene. Part 1. pp. 71-107.

This paper is a very excellent account of all the work that has been done on the subject of kala azar in the Mediterranean districts.

C. M. W.

GABBI (U.). *On the Identity of Infantile and Donovan's Leishmania (Kala Azar).—Il. Trop. Med. & Hyg.* 1913. July 1. Vol. 16. No. 13. pp. 198-199.

In this paper the author points out that the supposed differences, cultural and inoculative, between Indian and Mediterranean kala azar have been disproved by recent work.

C. M. W.

GABBI (U.). *Sulla Storia del Kala-azar del Mediterraneo.—Malaria e Malat. Paesi Caldi.* 1913. Apr.-May. Vol. 4. No. 3. pp. 198-202.

This paper continues the discussion between the author and LAVERAN on the first discovery of kala azar and its parasite in the districts on the Mediterranean (see this *Bulletin*, Vol. 1, p. 633). The author points out also that in the review of a paper by MAZZITELLI in Vol. 1 of this *Bulletin* it is not clearly stated that the first case of kala azar studied by the author (Gabbi) in Rome was one which had come from Messina. The author further regards as *inexact* the term "review" which was used in connection with his paper on the canine origin of kala azar summarised on page 632 of the same volume. He prefers to name his paper a "critical study."

C. M. W.

## CAUCASUS AND CHINA.

GURKO (A. G.). *Vier Fälle von Kala-Azar*. [Four Cases of Kala Azar.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. May 20. Vol. 74. No. 2. pp. 355-368. With 9 text-figs.

This paper deals with four cases of kala azar in the Caucasus district of south eastern Russia. The author states that only four cases have been so far recorded in Russia. Two of these were described by NIKIFOROW and PETROW and contracted the disease in middle Asia. The two remaining cases, noted by MARZINOWSKY and KALATSCHNIKOW, were from Russia and Siberia (the governments of Mogilew and Tobolsk). In the Caucasus region though oriental sore is common no cases of kala azar have been recorded. In the course of five months the author has met with the four following cases diagnosed by spleen puncture:—

1. Armenian child of six and a half years of age of the town of Eriwan.

2. A boy aged six years and nine months in Tiflis. The disease probably was contracted in Baku or some other southern town. Both these cases were treated by intravenous injections of salvarsan with distinct signs of improvement.

3. A youth aged sixteen, a labourer of Eriwan.

4. A Mohamedan youth aged nineteen from the Elisabethpol district.

A fifth case in a Mohamedan girl of sixteen years was seen but no puncture was made. All these were typical cases of kala azar and offered no peculiar features.

As the paper was passing through the press the author met with five further cases.

C. M. W.

HILL (R. A. P.). *Note on a New Sign in Kala Azar*.—*Lancet*. 1913. Aug. 9. p. 392.

It is pointed out that a form of "splenic anemia" in children and occasionally in young adults has long been known in Pekin. The cases all came from villages outside the city and were first shown by ASPLAND to be kala azar. The author has seen eleven cases in the last two years, only one over fourteen years of age; this was a girl about 20 who had been ill for a long time. Besides these there have been three adult cases in the wards but they were all from South China, two from Fukien and one from Honan. The author has diagnosed the cases several times by spleen puncture, but has not been able to find parasites in the glands as done by COCHRAN. In Pekin the diagnosis of the disease is rarely difficult.

The sign referred to in the title is the peculiar behaviour of kala azar blood when mixed with a special diluent which the author has used in making leucocyte counts in blood. When the blood is diluted, instead of the red cells disappearing at once as they do in other diseases (typhoid fever, typhus, relapsing fever, ankylostomiasis, pernicious anaemia, benign tertian malaria, hepatic cirrhosis) they run together into great lumps, which can only be broken up by long and vigorous shaking—in fact it is



usually impossible to make a count at all by this method. This reaction has occurred in every one of eight consecutive cases. In one other case the reaction occurred and in this it was impossible to exclude kala azar.

The diluting fluid used is the following:—

- A. Wright's modification of Leishman's stain,  
saturated and filtered ... .. 2 parts.  
Pure methyl alcohol ... .. 1 part.  
(This keeps indefinitely if well corked.)

- B. 0.1 per cent. solution of pure sodium chloride in distilled water.

For use add 1 part of A to 3 parts of B, shake well and use within half an hour. If a precipitate forms or the cells overstain add a little more methyl alcohol to A. Dilute the blood 1 in 20 or 25 and mix promptly. If the mixing is done too slowly lumps may form in the blood, but in kala azar it is impossible with the utmost expedition to avoid lumping. In one case in which the reaction was least marked the case recovered under big doses of quinine.

C. M. W.

#### KALA AZAR IN DOGS.

KOHL-YAKIMOFF (Nina), YAKIMOFF (W. L.), & SCHOKHOR (N. J.).  
Leishmaniose Canine à Tashkent.—*Bull. Soc. Path. Exot.*  
1913. June. Vol. 6. No. 6. pp. 432-433.

The authors point out that oriental sore is a common disease in Turkestan and that the first case of infantile kala azar found in this district was described by SLUKA and ZARFL in Vienna. After this discovery further cases were described by MARZINOWSKY in a child from Wolynsk, by NIKIFOROFF, from Tashkent, by KALAT-SCHINIKOFF from Tobolsk and by GOURKO in two infants and two adults from Tiflis.

In April 1913 the authors commenced an examination of the dogs of Tashkent. At the time of writing this paper they had examined 76 dogs, 22 of which were shown to be infected giving the very high percentage of 28.9 of dogs suffering from canine kala azar.

In the same month the authors saw a female child of nineteen months suffering from the disease.

C. M. W.

LAVÉLAN. Présentation d'un Chien atteint de Leishmaniose et de Kératite.—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 447-478.

A dog was exhibited by the author to show an opacity of the left cornea which had developed on July 8th after an inoculation into the saphenous vein of a culture of leishmania of Indian kala azar on June 10th. It would appear from this corneal lesion that the dog was infected though the author had so far been unable to discover leishmania. This is the second time the author has noted corneal opacity as a sequel to inoculation with virus of kala azar. On a former occasion a dog was inoculated

(June 3rd, 1909) intrahepatically with the virus of Tunisian kala azar. The dog became infected and had developed almost entire corneal opacity of both eyes by April 1910. In this case leishmania were found in the liver and spleen. NICOLLE working in Tunis has noted similar lesions in dogs on two occasions. It is pointed out that protozoal infections (leishmania, trypanosome, toxoplasma) all tend to produce this corneal opacity in dogs.

C. M. W.

In Tehran, where oriental sore is very prevalent, Dr. A. R. NELIGAN\* has discovered that the dogs suffer from ulcers about the face and that in these ulcers leishmania are often numerous. Examination of the internal organs of the dogs has not revealed a general infection, so that it would appear that the dogs were suffering from oriental sore, a disease which has not hitherto been found to occur naturally in these animals. This observation is of much interest in view of the fact that in the Mediterranean districts dogs may suffer from kala azar.

C. M. W.

#### TROPICAL SORE.

GONDER (Richard). *Experimentelle Übertragung von Orientbeule auf Mäuse*. [Experimental Transmission of Tropical Sore to Mice.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. June. Vol. 17. No. 12. pp. 397-403. With 2 plates.

The author has been able to infect mice both with *Leishmania infantum* and *L. tropica* by intraperitoneal and intravenous injections of the cultures in NNN medium. He points out that NICOLLE and his co-workers first showed that oriental sore could be produced in man, monkeys and dogs by the cutaneous inoculation either of virus from the sore or young cultures of *L. tropica*. LAVERAN has recently shown that old cultures of *L. tropica* will produce oriental sore in the monkey. Though there has been this success with the larger animals, and though infection of small laboratory animals has been produced by several workers by injection of cultures of *Leishmania infantum* of Mediterranean kala azar, no satisfactory infection of small animals has been produced by the injection of cultures of *Leishmania tropica*.

The materials used in these experiments were cultures of *Leishmania infantum* and *L. tropica* from Tunis. It was found, contrary to the generally accepted idea, that the cultures which contained a maximum of long flagellate forms produced infection better than old cultures which contained many round forms. To the liquid in each culture tube was added half to 1 cc. of physiological saline solution and 1 cc. of such mixture was injected into each mouse of 20 grams weight. It was noted that unless the saline was added the culture fluid frequently produced serious symptoms of toxic poisoning with dark red brown urine. The

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\* *Jl. Trop. Med. & Hyg.* 1913. May 15. Vol. 16. No. 10. p. 156 [Annotation].

course of the infection could be followed by the operation of liver puncture which was very well tolerated by the mice. In both cases (*L. infantum* and *L. tropica*) a general infection was produced and the incubation periods were identical, enlargement of the liver and spleen being seen in about one month. In the case of *L. tropica* infection some of the mice ultimately, in several months, developed cutaneous ulcerations of varying severity. After intraperitoneal injection of cultures of *L. tropica* liver puncture revealed parasites in one mouse after one month, in two mice after two months and in other mice after three months. There was a morphological difference in the parasites first recovered from the liver. In the case of *L. tropica* the average size was  $5.7 \mu \times 2.4 \mu$  while the trophonucleus was irregular in shape and less compact in structure. With *L. infantum* the first parasites found in the liver had an average size of  $1.3 \mu \times 1.5-4.5 \mu$ . By the use of wet fixation in sublimate alcohol the author has been able to demonstrate in some parasites a short flagellum either representing the retraction of one already there or the commencement of the formation of a new one. He believes that by an intensive study of the parasites it would be possible to distinguish between them morphologically.

The great interest in this investigation lies in the results of the *L. tropica* infections. Internally, parasites only occurred in the liver and spleen, never in the bone marrow nor any other organ. The infected mice with enlarged liver and spleen appeared for some months to be quite healthy and undisturbed in any way by the leishmania. One mouse after four months became ill and died and enormous numbers of parasites were found in the greatly enlarged liver and spleen. Later another mouse died in a similar manner. Two other mice had no enlargement of the liver and spleen though these organs contained leishmania, so that in these animals the disease varies very much in its severity. In four other mice after a lapse of four months there developed oedematous swellings of the feet. Puncture of these produced a clear serous fluid which was practically a pure culture of *L. tropica* both free and included in leucocytes and lymphocytes. After some days the swellings commenced to necrose and similar lesions developed in the ears and tail and in one a necrosis of the lower jaw. In all these lesions *L. tropica* occurred. Two of these mice had a heavy infection of the liver and spleen till the peripheral lesions developed whereupon the parasites in the liver and spleen became very scanty. Another mouse which had extensive ulceration of the paws, tail and ears had parasites internally only in the liver. The fourth mouse, also with extensive ulceration had very large liver and spleen, in both of which as well as in the ulcers very numerous parasites occurred.

The author has been so far unable to produce infection by the cutaneous or subcutaneous injection of cultures of *L. tropica*. By the intraperitoneal and intravenous injection of material from the liver, spleen or ulcers of infected mice infection of the liver has been produced after an incubation of five to six weeks, though up to the present skin ulceration has not appeared.

In only one case has an oriental sore been produced, in the hind foot, by the subcutaneous injection of extracts of infected organs of mice.

The author naturally wonders whether the course of *L. tropica* infection in mice is not similar to that in man and suggests that possibly an infection of the liver and spleen occurs unrecognised before the cutaneous lesions develop. The long incubation period in man speaks in favour of this view.

C. M. W.

**SOCIETÀ ITALIANA FRA I CULTORI DELLA MALATTIE ESOTICHE.**

**Riunione Privata tenuta a Messina il 15 Giugno 1913 intorno alla Leishmaniosi Umana in Italia.** Atti, Relazioni, Comunicazioni Scientifiche (con tavole) per cura del Prof. Dr. G. Spagnolio, Dr. M. Signer, Segretari della Riunione.—180 pp. With 5 plates and 4 maps. 1913. Messina: Stab. Tipografico Guerriera. [Tropical Sore. pp. 153-170.]

- i. LA CAVA (F.). **Sulla Frequenza, Diffusione e Sindrome della Leishmaniosi della Cute e delle Mucose (L. esterna) nell'Italia inferiore.** pp. 153-160.

This is an introductory address on the subject of tropical sore and reviews the present position of the subject. The disease is wide spread in the extreme south of Italy and Sicily, many cases having been now identified. Four cases of involvement of the mucosa of the mouth and nose have been recorded in Italy and Sicily—three by the author and one by PULVIRENTI. A very interesting case came under the author's notice in which a child suffered from this disease and had a large spleen and fever while three other older children of the same family had been proved to suffer from infantile kala azar. Spleen puncture was performed on the child with the sore but no leishmania could be discovered, so that the fever and splenic enlargement remained unexplained unless it be assumed that the oriental sore had given a partial protection against the more serious disease.

- ii. PULVIRENTI (G.). **Anatomia Patologica, Diagnosi, Prognosi e Cura.** pp. 160-168.

A review of past work.

- iii. FAZZARI (G. B.). **Un Caso di Bottone d'Oriente in Antonimina (Reggio Ca.).** pp. 169-170: also *Malaria e Malat. d. Paesi Caldi*. 1913. June-July. Vol. 4. No. 4. pp. 264-265. With 1 text-fig.

A case of oriental sore on the right side of the upper lip in a woman aged 38 years from Antonimina in Southern Italy. Diagnosis was made by discovery of numerous leishmania.

C. M. W.

SANT 'ANNA (Firmino). *Trabalhos experimentaes sobre um Caso de Leishmaniasse de Origem Brasileira*. [Experimental Researches on a Case of Leishmaniasis of Brazilian Origin.]—*Medicina Contemporanea*. 1913. Aug. 24. Vol. 31. No. 34. pp. 267-272. With 2 text-figs.

From a case of cutaneous leishmaniasis from the Upper Amazon the author has been able to infect monkeys. A *Cercopithecus* was inoculated with a saline emulsion of scrapings from the sore into the subcutaneous tissue in the supraorbital region and also by scarification on the skin of the forearm. Sixty-six days after inoculation a small ulcer was noted over the eye and leishmania in small numbers were discovered in the lesion. Five days later part of the lesion was excised for histological study and further inoculation. The lesion increased in size for two months till a large ulcer covering the supraorbital region was developed. The scarification on the arm did not lead to any appreciable result.

Another monkey (*Cercopithecus*), inoculated as the first, developed a small ulcer in the supraorbital region in which leishmania were found. This lesion only persisted for about one and a half months. The arm inoculation as in the first monkey produced no result. A third monkey was inoculated in the lower margin of the nares, with material from the excised sore of the first monkey. Nine days later there was a small excoriation covered with crust. This soon disappeared, but two months later there occurred at the same spot a nodule in which leishmania were found. A *Cynocephalus* was also inoculated with material obtained from the first monkey on the ears. Twenty-five days later there was a nodule at this spot. It increased in size and ulcerated. Smears contained many mononuclear cells as in the lesions on the other monkeys, but parasites were not discovered.

The greater part of the paper is occupied with a description of the leishmania and the histological study of the lesions.

C. M. W.

BOLETIM DA SOCIEDADE BRASILEIRA DE DERMATOLOGIA. 1912. Vol. 1. Nos. 1-2-3. 80 pp. With 10 plates. [Resumé in French. pp. 59-80.]

The publication under review contains an account of the proceedings of the Dermatological Society of Brazil. During the year several cases of leishmaniasis of the skin and mucosae were shown at the society's meetings. In some of these cases the disease was limited to the skin as in oriental sore, while in others there was extensive involvement of the nasal, pharyngeal and buccal mucosae producing the condition which has been previously described by SPLENDORE and others (see this *Bulletin* Vol. 1, p. 11). In the discussions upon the case many of those present were familiar with the disease, which must have a fairly wide distribution in Brazil.

C. M. W.

MARTOGLIO (F.). *Il Bottone Orientale in Abissinia*.—Extract from vol. "In Onore del Prof. Angelo Celli nel 25 Anno di Insegnamento." 1912. pp. 411-419. With 1 text-fig. and 1 plate. [Rome: Tipografia Nazionale di G. Bertero.]

This paper contains a description of three cases of oriental sore from Abyssinia. A plate is given showing the typical leishmania found in the lesions. The author concludes that the disease is endemic in Abyssinia, including Eritrea. Though the diagnosis has not been made by spleen puncture the author believes that kala azar exists here also, especially in the provinces of Barentu and Agordal as it does in the neighbouring provinces of the Sudan.

This is the first time that oriental sore has been recorded from Abyssinia.

C. M. W.

UFFERTE (L.) & PELLIER (J.). *Sur un Cas de Bouton d'Orient (Clou de Gafsa)*.—*Ann. de Dermatologie et Syphiligraphie*. 1913. June. Vol. 4. No. 6. pp. 331-334. With 1 text-fig.

One of the writers contracted oriental sore while on military duty in North Africa. He stayed in Gafsa for two days and during this time he wore no gloves and had many slight abrasions of the skin. Fifteen days later without any surrounding inflammation a wart began to develop on the back of each hand. Caustic applications were of no avail and only transformed the lesions into indolent ulcers. Eventually, seven months after their first development, leishmania were found. The authors believe that the disease was contracted at Gafsa, from which town the disease has received one of its names (*clou de Gafsa*).

C. M. W.

In the *Journal of Tropical Medicine & Hygiene*\* it is stated that Dr. MINETT has found leishmania in the skin disease known as "forest yaws" in British Guiana. FLE has previously found leishmania in this disease in both French and Dutch Guiana [see *Kala Azar Bulletin* No. 2, pp. 108-109].

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\* *Jl. Trop. Med. & Hygiene*. 1913. Aug. 1. Vol. 16. No. 15. p. 240 [Notes & News].

## SLEEPING SICKNESS.

BERNARD. Relation d'une Tournée Médicale faite dans la Région du Djéma et du M'Bomou (Oubangui-Chari) Avril et Mai 1912. —*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 321-332.

Sleeping Sickness was first recognised at Zémio in 1903 and its appearance corresponded with the erection of the factory at Djéma where it was brought by the company's labourers. From Djéma the disease has spread in all directions. This is a striking instance of the part played by infected human beings in the spread of the disease through their removal to distant parts.

The author writes that he cannot pass over in silence the part played by the native concubines of Europeans in the spread of sleeping sickness. He recognised trypanosomes in the glands of two of these women who had been brought from clean districts and who had without doubt been recently infected in the neighbourhood of the villages of the labourers and had thus become intermediaries for the transmission of the disease to the Europeans.

Among the clinical signs of the disease the authors lay stress on the alteration in the genital organs: impotence in the male, and amenorrhoea or dysmenorrhoea in the female. The signs are not constant, but they are extremely early and the same is the case with gastro-intestinal disturbances. The mortality from sleeping sickness in this region has been considerable.

A brief account is given of the various methods of prophylaxis which have been devised to deal with the disease.

W. Yorke.

BOURRET. Recherches sur le Parasitisme Intestinal, la Dysenterie et la Maladie du Sommeil à Saint Louis Sénégal.—*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 283-307.

The discovery of trypanosomes by direct examination of a drop of blood from sleeping sickness patients is rare in the Sénégal; the author only found trypanosomes in one case out of 45 and of these 20 were untreated. In the French Congo on the contrary MARTIN, LEBOEUF and ROUBAUD found trypanosomes in the blood of 36-45 per cent. of cases, whilst the English Commission to the Belgian Congo found the parasites in 13·6 per cent. of cases.

In view of the discovery of *T. rhodesiense* it was of interest to ascertain whether the human trypanosome of Sénégal was really *T. gambiense*. On examination this proved to be the case; no posterior nuclear varieties were encountered. The pathogenicity of the parasite was then examined. A guinea-pig died 6 months after inoculation; in rats the incubation period was 12-14 days and the duration 89-185 days; one single mouse died on the 35th day of the disease. A horse showed trypanosomes in its blood for the first time 46 days after inoculation. Its blood was not virulent for *Cercopithecus patas* 7 months after inoculation, but it had not acquired immunity, for 4½ months after a second inoculation of the Sénégal virus its blood was infective for monkeys and the animal had become emaciated. A sheep

had febrile attacks and showed signs of weakness a month after inoculation, although trypanosomes were never seen in its blood for a year; nevertheless at the end of this time it was infective for monkeys. A month after a second inoculation trypanosomes were found in its blood. The animal died 15 months after the first inoculation. Another sheep inoculated in Paris has never shown parasites in its blood nor has it presented signs of disease. Its blood, however, was virulent for white rats and its serum was protective not only against the Sénégal virus, but also against *T. gambiense*. In 17 *Cercopithecus patas* the incubation period varied from 9-35 days. As a rule, the animals did not live more than six months.

*The Wassermann reaction in sleeping sickness.*—Not having at his disposal any satisfactory extract of syphilitic liver, the author used as antigen an alcohol extract of normal human heart. As a result of his observations Bourret comes to the conclusion that the Wassermann reaction as carried out by him with non-specific antigen and the serum of sleeping sickness patients is too inconstant, at least in the second stage of the disease, to be of any value as a means of diagnosis.

#### *Treatment of sleeping sickness.*

(1.) *Atoxyl-orpiment.*—These drugs were administered as recommended by THIROUX: two series of 20 days of treatment separated by a period of rest of 8 days, each series comprising 5 hypodermic injections of atoxyl (.5 gm.-.75 gm.) at 4 day intervals and the daily administration of pills of orpiment (commencing at .3 gm. and increasing each day by .1 gm. to 1.1 gm. in those cases which tolerate the latter dose). As a rule this treatment was well borne. All the patients thus treated were in the second stage; none were cured, but in almost all the condition was ameliorated. The results in 12 patients so treated are given in a table.

(2.) *Atoxyl-emetic.*—Two periods of treatment separated by an interval of a month, each comprising 3 subcutaneous injections of atoxyl (.5 gm., .75 gm. and 1.2 gm. or 5 gm., 1 gm. and 1.5 gm. according to the weight), and afterwards from the 5th to the 15th day 10 daily injections of emetic in doses varying from .09 gm. to .15 gm. according to the weight. The results of treating 6 patients by this method are given in a table; they are very similar to those obtained by the previous method.

(3.) *Arsenobenzol.*—This drug has been used in 4 cases. It was always given intravenously. The injections in the first 3 cases were .55 gm. to .6 gm. at intervals of a month, in the 4th case the dose was .5 gm. at intervals of 8 days and 4 doses were given instead of 2 or 3 as in the previous cases; the treatment was well borne. The results which are given in a table are not very satisfactory.

In conclusion the author writes that from the comparatively few cases treated it would not seem that arsenobenzol ought to displace the earlier remedies in the therapy of sleeping sickness. The atoxyl emetic combination appeared to give the best results.



LURZ (R.). Ein Mittel von L. Brieger und M. Krause zur Behandlung der Trypanosomen im menschlichen Organismus. [A Remedy of L. Brieger and M. Krause for the Treatment of Human Trypanosomiasis.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Sept. Vol. 17. No. 18. pp. 636-640.

This paper records the results in German East Africa of treating a number of sleeping sickness patients with the remedy of BRIEGER and KRAUSE. The following summary is given:—

Doses of .25 gm. of the drug given after food [in cachet] twice daily resulted in all ten cases in severe diarrhoea, so that the patients became emaciated and subsequently refused to take the drug. Trypanosomes were found in the blood of all the patients on the 14th day of treatment.

When given as follows the drug was tolerated by six patients:—1 gm. in the first week, twice as much in the second and third weeks and three times as much in the fourth and fifth weeks and .25 gm. in the sixth week. One patient became markedly emaciated during treatment. Trypanosomes were found in all the patients in the gland juice during and after treatment and the condition was not ameliorated.

Seven other patients bore .1 gm. in the first ten days, .2 gm. on the next ten days and .3 gm. on the following 22 days. In three female patients the general condition became so bad that atoxyl treatment had to be resorted to. In one case the glands diminished in size and the trypanosomes disappeared from the gland juice, but not from the blood. The condition of the remaining patients was not altered.

In conclusion the author writes that this drug, either in large or small doses, is valueless for the treatment of human trypanosomiasis.

W. Y.

MORGENROTH (J.) & TUGENDREICH (J.). Aethylhydrocuprein und Salicylsäure als Adjuvantien des Salvarsan. — *Berlin. Klin. Wochenschr.* 1913. No. 26. 16 pp.

The gist of this paper has been published previously (see this *Bulletin*, Vol. 2, p. 248). Details of the results of treating mice infected with nagana with these drugs alone and in combination are given in thirteen tables.

W. Y.

RECAMIER. Un Cas de Trypanosomiase observé chez un Tirailleur indigène à Fort-Lamy.—*Ann. d'Hyg. et Méd.-Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 442-443.

A clinical account is given of a case of sleeping sickness in a native trooper from Fort-Lamy. There is nothing noteworthy to record. Quinine had no influence on the temperature and the case ended fatally.

W. Y.

#### TRANSMISSION EXPERIMENTS.

KLEINE (F. K.) & FISCHER (W.). Schlafkrankheit und Tsetsefliegen. \* (II. Mitteilung.) [Sleeping Sickness and Tsetse Flies.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. Aug. 27. Vol. 75. No. 2. pp. 375-382.

Sleeping sickness due to *T. gambiense* is limited in East Africa to the localities in which *G. palpalis* occurs. In their earlier

\* The first portion was summarised in this *Bulletin*. Vol. I. p. 270.

papers the authors left undecided the question as to whether this fact depended on climatic conditions or whether it was because this particular tsetse was the most suitable host of the infection. The practical application of this question lies in the fact that, if temperature and humidity be determinative, similar climatic conditions are equally likely to play a part in those regions where *G. palpalis* does not exist, but only the widely spread *G. morsitans*; if, however, *G. palpalis* itself is the chief factor, the infection will be limited to the distribution of this fly.

In order to decide this point a series of closely parallel experiments were undertaken with laboratory bred *G. palpalis* and *G. morsitans* in the neighbourhood of the Rutschugi station about six hours north of the great caravan route between Tabora and Ujiji. The climate was hot in the daytime and cold at nights, and was thus quite different from that of sleeping sickness districts. The investigations were undertaken in the rainy season.

Thirteen experiments were performed and in each a different strain of *T. gambiense* was used, monkeys (*Cercopithecus rufo-iridis*) being infected from various sleeping sickness patients. Laboratory bred flies of both species were fed on the infected monkeys at the same time, or one immediately after the other, on each of four days, and then as usual on healthy animals. When one of the monkeys became ill the flies living were divided into small groups and these were fed on different animals. In this way a rough idea of the number of infective flies was obtained. After feeding each group the surviving flies were killed and examined. Details are given of the thirteen experiments and the results are set forth in two tables. Of 881 *G. palpalis* only 8 became infective, whilst developmental forms were found in 15 others which died or were killed after the 12th day of the experiment. The developmental period of the trypanosome in the fly was 28 to 30 days. In regions the climate of which is favourable to the development of the trypanosome, that is in sleeping sickness districts, 2.5 to 6 per cent. of the *G. palpalis* became infective after an interval of only 20 to 25 days.

Of the 881 *G. morsitans* fourteen became infective and developmental forms were observed in 40 others which died or were killed after the 12th day of the experiment; the developmental period was 37 to 42 days, which is thus on average 10 days longer than the corresponding period in the case of *G. palpalis*.

As a result of these experiments it appears that the connection of sleeping sickness and *G. palpalis* is dependent not on the specificity of the tsetse, but on the climatic conditions obtaining on the lake shores, which are peculiarly favourable to the development of the trypanosome. Furthermore, it seems that *G. morsitans* might transmit *T. gambiense* in places,—e.g. the highlands,—which have hitherto been considered safe from the infection.

Incidentally it might be remarked that it is known that in spite of suitable external conditions the infection very frequently does not spread. In these cases the trypanosomes have apparently lost their capacity for development in *Glossina*. The reason for this is not clear, but as a matter of fact the authors have noted

strains of trypanosomes with which it is impossible—or possible only with great difficulty—to render *Glossina* infective.

[This paper should be compared with those of KINGHORN and YORKE on the influence of climatic conditions on the development of *T. rhodesiense* in *G. morsitans* (see this *Bulletin*, Vol. 1, p. 43 and 126).]

W. Y.

#### WILD ANIMALS AND SLEEPING SICKNESS.

AUSTEN (E. E.). *The Present Position of the Problem of Big Game, Tsetse Flies, and Sleeping Sickness.*—*Jl. Soc. for Preservation of Wild Fauna of the Empire*. 1913. Vol. 6. pp. 57-71.

The first portion of this paper summarises our knowledge of the relation of sleeping sickness to tsetse flies and big game, whilst the latter portion is a reply to the arguments developed by YORKE in lectures before the Zoological Society and elsewhere on the relationship of the African fauna to the spread of sleeping sickness.

With reference to the experiment advocated by YORKE (see this *Bulletin*, Vol. 2, p. 238) the author writes "Suffice it to say that the suggested experiment is devised on scientific lines, and that, provided it be possible to ensure the elimination of all contingencies by which the results might conceivably be vitiated, no valid objection can be urged against its being carried into effect."

The possibility of there being other 'Reservoirs' of *T. rhodesiense* than big game is considered. Should this be found to be so the author is of opinion that the case against the game might be materially affected. On the other hand, should it be shown that the parasite sometimes occurs in the blood of certain small mammals without doing these animals any harm, big game might still be by far the more important reservoir, since, owing to the great disparity in size of the animals, the total mass of living antelope blood in any area must usually be much greater than the total mass of living small mammal blood within the same limits.

There is yet another possibility, namely, that certain human beings may be reservoirs of the parasite. The possibility of the existence of human reservoirs in the case of *T. gambiense* is admitted by some authorities. The author quotes a recent paper by TOMP (see this *Bulletin*, Vol. 2, p. 255) in support of this view. Owing to the greater virulence of *T. rhodesiense* it must be admitted that at first sight it would seem less likely that human beings should be reservoirs of it than of the parasite just mentioned. KINGHORN and YORKE write, however, that "occasionally a patient is more resistant, and one native definitely proved to be infected with *T. rhodesiense* is still alive and in a state of apparent good health a year later." To what extent mankind is a reservoir of this parasite remains to be proved. It is possible that in districts infested by *G. morsitans* the ratio of human reservoirs to population is extremely high. According to KINGHORN and YORKE in North Eastern Rhodesia only about 1, in 500 (a percentage of 0.2) is naturally infective with *T. rhodesiense*. Evidence on this head with regard to Nyasaland is as yet very scanty, but, writing on June 7, 1912,

Sir David BRUCE stated that he and his colleagues had found this particular parasite in 8 out of 38 experiments, amounting to a percentage of 21. This would appear to indicate a state of things much more serious than that which apparently obtains in North-Eastern Rhodesia; and, if anything like so high a percentage as 21 of wild *G. morsitans* in Nyasaland is naturally infected with *T. rhodesiense* (or *brucei*), it is difficult to understand the comparatively trifling incidence of sleeping sickness among the population of that country, as at present reported, except upon the assumption that the majority of the people are immune. [The author has made a remarkable mistake in stating that the Royal Society Commission found *T. rhodesiense* in 21 per cent. of wild *G. morsitans*. He has apparently overlooked the fact that in each of the 38 experiments performed by this Commission many flies were used. As a matter of fact, BRUCE and his colleagues found that of 10,000 wild *Glossina morsitans* examined by them only .2 per cent. were naturally infective—a figure which is identical with that of KINGHORN and YORKE. It is necessary to draw attention to this error, as it is apparently on the misconception that 21 per cent. of wild *G. morsitans* are infective that the author assumes "that the majority of the population are immune." Even assuming that a proportion—which must be small in view of the rapidity with which the great majority of the recognised cases of the disease have died—of human beings are tolerant of the trypanosome and serve as carriers for long periods of time, these form in the reviewer's judgment a quite insignificant reservoir of the virus compared with the antelope.]

W. Y.

CORYNDON (R. T.). *Tsetse Fly and Big Game*.—*Jl. Soc. for Preservation of Wild Fauna of the Empire*. 1913. Vol. 6. pp. 41-56.

In this article the connection between tsetse fly and big game is discussed at considerable length. In conclusion the author writes: "It is not denied that fly is generally found with game, and in fact often follows it, but only that slaughter of game should not be authorised till it is shown that by killing the game you will do away with nagana disease, not only with *Glossina morsitans*. I desire to make out a common-sense case against the petrified belief that fly is dependent upon game, and so to prevent, or at least to postpone, the slaughter, untried and certainly unconvicted, of many thousands of beautiful and valuable animals."

W. Y.

T. BRUCE.

FISCHER (W.). *Über das Vorkommen von Kernverlagerungen bei Trypanosoma brucei*. [On the Occurrence of Aberrant Nuclei in *Trypanosoma brucei*.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Sept. Vol. 17. No. 18. pp. 621-626. With 16 text-figs. and 1 curve.

The author refers to the statement of KINGHORN and YORKE that 16 per cent. of the wild game of the Luangwa Valley were

infected with the human trypanosome. The correctness of these observations, however, appears doubtful, as KINGHORN and YORKE did not record the existence of *T. brucei* which is closely related to *T. rhodesiense*.

Trypanosome strains were examined in a sleeping sickness free area as regards forms exhibiting change of position of the nucleus, with the idea that *T. brucei* would also be found to show posterior nuclear forms. The work was undertaken at Malai, between Tabora and Ujiji, which is absolutely tsetse fly free. Wild *Glossina morsitans* were caught in the surrounding fly area and brought to the laboratory and fed on dogs. In this manner six separate strains, morphologically and biologically identical, were obtained. An account is given of the pathogenicity and morphology of the strains, which were found to be identical with those of the parasite which one was accustomed to regard as *T. brucei*. Recently it has been shown that the original *T. brucei* is monomorphic and not dimorphic. It is possible that this is due to long maintenance by passage through laboratory animals, but in any case the trypanosome which is found in Central Africa in *morsitans* regions and which possesses all the biological characters of *T. brucei* is of a definitely dimorphic type.

Forms exhibiting aberrant nuclei were most commonly met with in guinea-pigs (21.3 per cent.) and more rarely in dogs and monkeys (3.5 per cent.).

In addition to the above six strains posterior nuclear forms were found in a blood film from an infected mule. KLEINE observed posterior nuclear forms in preparations from animals infected with *T. brucei* on the east coast of Lake Victoria, and ECKARD in the blood of a rat inoculated from a bushbuck from Tanganyika.

A biometric curve constructed by measuring 100 trypanosomes on each of ten consecutive days of the infection in a monkey is given.

In the author's opinion it is not permissible to refer to the trypanosomes of game on account of certain morphological peculiarities as *T. rhodesiense*. He holds the same view as TAUTE (see this *Bulletin*, Vol. 2, p. 348) that the assumption of KINGHORN and YORKE that 16 per cent. of the game of the Luangwa Valley is infected with *T. rhodesiense* still requires proof. [The author has brought forward no evidence to show that *T. brucei* (Uganda) of BRUCE, or *T. ugandae* of STEPHENS and BLACKLOCK, is different from *T. rhodesiense*. It is highly probable that these parasites are identical.]

W. Y.

HECKENBOTH (F.) & BLANCHARD (M.). Réaction de Fixation, en Présence d'Antigène Syphilitique, dans la Syphilis, le Pian, la Trypanosomiase et l'Ulcère phagédénique au Congo français.—*Compt. Rend. Acad. Sciences*. 1913. Sept. 1. Vol. 157. No. 9. pp. 437-440; and *Caducée*. 1913. Sept. 20. Vol. 13. No. 18. p. 244.

The method used was that devised by LEVADITI and LATAPIE, which is characterised by the employment of alcoholic extract

of the liver of a syphilitic foetus as antigen and the anti-sheep complement and amboceptor normally found in almost all human sera. This simplified method is valuable for use in the Colonies; the author found that such a haemolytic system is complete in the majority of sera and that it was only necessary to use guinea-pig complement and anti-sheep amboceptor of the rabbit in about 3 per cent. of cases.

Experiments on the fixation of complement were performed with the serum of 73 cases of trypanosomiasis, of which 4 showed well-marked lesions of syphilis. Deducting these cases the following results were noted:—Of 27 cases in good condition 15 gave a positive reaction (55 per cent.), and of 41 patients in bad condition 27 gave a positive reaction (65 per cent.). There is hence no doubt that syphilitic antigen can fix complement with trypanosomal amboceptor in more than half the cases whatever may be the state of the patient.

It was not possible to determine why the reaction was positive with the serum of certain patients only.

W. Y.

ROSENTHAL (Felix). *Untersuchungen über die Genese des Rezidivs bei der experimentellen Trypanosomeninfektion.* [Investigations on the Genesis of Relapses in Experimental Trypanosomiasis.] — *Zeitschr. f. Hyg. u. Infektionskr.* 1913. June 26. Vol. 74. No. 3. pp. 489-538.

The following summary is given:—

The experiments described show that the trypanocidal measure of a specific trypanosome immune serum does not represent the chief factor in the conversion of a normal strain into a relapse strain. It seems probable that besides the trypanocidal antibodies independent serum substances exist—called by the author relapse bodies—in response to which the trypanosomes are stimulated to the formation of receptors differing from those of the parent strain. The following observations support the view that relapse bodies exist as entities apart from trypanocidal immune bodies.

1. The dissonance between the trypanocidal power of trypanosome immune serum and its relapse strain formation power as observed *in vitro*.

2. The greater thermolability of the relapse strain formation bodies which are destroyed by heating to 60° C. for 15 minutes, whilst the trypanocidal antibodies remain intact.

3. The relapse strain formation properties of specific trypanosome immune sera appear to be bound in preference to the albumen fraction of the serum, whilst the trypanocidal bodies are found to be as much associated with the globulin as the albumin fraction.

According to the amount of trypanocidal substances and relapse bodies in immune sera the following possibilities were noted on treatment of normal trypanosomes in the test tube with sufficient but not lethal quantities of the immune serum.

1. The trypanosomes become relapse parasites without delay.

2. After more or less delay of the infection the surviving trypanosomes possess relapse strain properties.

3. The trypanosomes succeeded after a distinct or considerable time in developing again into normal parasites.

4. The trypanosomes may exhibit normal strain and relapse strain properties at the same time.

Acquired relapse strain properties need not be permanent. The return of the relapse strain to the original strain need not depend, however, on the return of all the parasites, but can be accomplished by the existence of true mutation forms due to the overgrowth of trypanosomes of the variation type which retrogress to the normal strain.

Biological distinctions exist between the relapse strain produced through contact with immune serum in the test tube and that resulting spontaneously in the course of infection. In the same way relapse strains, produced by the action *in vitro* of immune sera of different days, exhibit biological differences as tested by the immunity reaction.

Mice immune serum shows *in vitro* little or no trypanolytic capacity; on the other hand the blood of cured mice possesses *in vivo* strong trypanolytic action. This is in agreement with the absence of haemolytic complement in mouse serum, whilst *in vivo* complement is demonstrable.

W. Y.

RAVENNA (Ettore). **Lesioni Endocardiche nella Tripanosomiasi Sperimentale.** [Endocardiac lesions in experimental trypanosomiasis.] — *Archivio per le Scienze Mediche.* 1913. July 10. Vol. 37. No. 3. pp. 236-249.

The author refers to a previous paper in which he published as a preliminary note some observations upon endocardiac lesions in dogs infected with trypanosomiasis. In the present paper he gives an account of further observations. The changes consist in the formation of small nodules of inflammatory nature on the valves, "miliariform endocarditis of BANTI." No trypanosomes were found in such nodules in any of the dogs examined. In one group of dogs examined the endocardiac lesions were found in seven out of ten. A control did not show such changes. The author concludes that in experimental trypanosomiasis in the dog alterations of the endocardium are frequent.

W. Y.

## PROTOZOOLOGY.

LAVERAN (A.) & FRANCHINI (G.). Infections expérimentales de la Souris par *Herpetomonas ctenocephali*.—*Compt. Rend. Acad. Sciences*. 1913. Sept. 1. Vol. 157. No. 9. pp. 423-426.

The authors give a brief review of the work on the flagellates of cat and dog fleas by PATTON, MARZOCCHI, NÖLLER and FANTHAM, and point out that the number of insects infected varies with the locality. They recall that the *Herpetomonas* possesses oval or rounded non-flagellate forms and flagellate stages, whose average body measurement is 10  $\mu$  with a flagellum about 8  $\mu$ .

Four white mice were inoculated intraperitoneally with gut contents of dog fleas containing herpetomonads. After 2, 3 and 4 days, parasites were found in the peritoneal exudate; in two, parasites occurred in the blood, the infection lasting 42 and 61 days respectively. A mouse subinoculated with peritoneal exudate showed parasites two days later, but the infection only lasted 48 hours. Another mouse, inoculated with blood showed parasites three days later, and infection lasted 52 days. In every case, parasites were rare or very rare. The parasites were usually spherical or oval. They are almost always free and the greater number are leishmania-like. Larger elements may occur in the spleen. Flagellate forms were very uncommon in the infected mice and occurred only in the blood.

The authors state that the parasites found in inoculated mice are very near to those of *H. ctenocephali*, the rarity of the flagellated elements constituting the chief difference. They also suggest that the parasite, if acclimatised to the new medium (mouse blood), might be able to become pathogenic, thus opening new ideas on the etiology of Leishmaniasis. The possible parasitic effects of *H. ctenocephali* must also be taken into account in attempts at transmitting kala azar by inoculating mice with contents of dog fleas.

A. Porter.

WENYON (C. M.). Observations on *Herpetomonas muscae domesticae* and some Allied Flagellates. With special Reference to the Structure of their Nuclei.—*Arch. f. Protistenkunde*. 1913. Sept. 25. Vol. 31. No. 1. pp. 1-36. With 3 plates, 6 text-figs., and 1 diagram.

The author examined houseflies obtained in Aleppo, Syria, using wet fixation and iron haematoxylin staining. The majority of the Aleppo flies contained the large *Herpetomonas muscae domesticae*. Smaller leptomonas forms were also present and the author is "convinced that they are merely stages of the larger flagellate, for transition forms connecting the two types are common." Some trypaniform flagellates also were found. Other preparations showed that "*Herpetomonas muscae domesticae* (Prowasek's supposed biflagellate) likewise has a trypanosome stage." Thus "the *Leptomonas* of ROUBAUD and others is merely a not actively dividing *Herpetomonas muscae domesticae* and (that)



both may pass through a transition into flagellates of a trypanosome type." The types of production of "small, round forms" [post-flagellates] are described. (1) If derived from leptomonas forms, cysts of the usual type are produced. (2) If the flagellate is trypaniform, more of the flagellum remains intra-corporeal and the body itself becomes rounded after approximation of its ends. (3) The body "simply contracts to a globular shape." Nuclear multiplication may occur during encystment.

Further examination of the flagellates of the Aleppo housefly has strengthened the opinion of the author that the flagellate of the housefly is not biflagellate but "that the forms showing the two flagella were the result of the precocious formation of new flagella in anticipation of a coming longitudinal division." The division of the basal granule of the flagellum, the karyosome of the kintonucleus and the division of the trophonucleus have been traced. The author believes that a cytosome and cytopharynx are present, using as an argument the presence of bipolar bodies which he believes are bacteria taken up by the *Herpetomonas*, which "renders the presence of a cytostome very probable."

The cytology of the trophonucleus is discussed in detail. "It is probable that the karyosome is built of three essential portions or substances. There is the chromatin material, the centriole, and finally a plastin material which binds the two former together." Full details of the division of the centriole are given, but it is clear that great variation in nuclear appearances results from the degree of abstraction of the stain. The division of the karyosome of the kinto-nucleus also is described. The basal granule is considered to function as a centrosome in nuclear division. The flagellum has an ectoplasmic sheath. In dividing forms it is considered that the flagellum of one daughter individual arises by the growth of a new rhizoplast near the old one. No axostyle structure of as definite a form as PROWAZEK describes could be found.

The author makes some statements regarding nomenclature. From what he has found among the flagellates of houseflies he believes that "there are four types which these [insect] flagellates may assume." It should be mentioned that stained preparations were used throughout. The four forms are designated Trypanosome, Crithidia, Leptomonas and Leishmania, and the author criticises the work of other authors on various Herpetomonads, as he considers their organisms have no trypanosome phase, which he has now put forward as essential to Herpetomonas. [But it must be remembered that information as to stages in the life cycle of any flagellate and the relation of intermediate forms can only be definitely decided by study of the living organism and actual watching of the processes of transition. Also, stained preparations alone cannot decide the question of mixed infections, which the above work has somewhat overlooked.]

The nuclear structure of certain other flagellates is briefly noted. The nucleus of *Trypanosoma rhodesiense* has a granule representing the centriole or the centriole together with chromatin. As previous workers have found, there are no definite

chromosomes in nuclear division. The author states that "Another feature brought out in [the] degeneration products of these trypanosomes is the structure of the flagellum," and he considers that in dividing forms the new flagellum arises *ab initio*, as in his *H. muscae domesticae*. [But can reliance be placed on features observed in admittedly degenerating forms, especially when stained material only is used?] *T. lewisi* was examined but little new found. Cultures of a *Leptomonas* from *Pulex irritans* on NNN medium gave many dividing forms, whose kinetonuclei are like those of the other flagellates. The author has the same ideas with regard to the origin of the daughter flagella as he has for *H. muscae domesticae*. A second description of *Cercomonas longicauda* is given. It is apparently the same organism as described by HARTMANN and CHAGAS as *C. parva*, but they regard the second flagellum as passing through the body, WENYON as passing over it, and the latter found neither axostyle nor rhizoplast as described by the German workers. Three plates illustrate the paper, but it is regretted that the magnification of the figures is not given.

A. P.

VON PROWAZEK (S.). **Notiz zur *Herpetomonas*—Morphologie sowie Bemerkung zu der Arbeit von Wenyon.** [Note on *Herpetomonas*—Morphology and Remarks on Wenyon's Paper.]—*Arch. f. Protistenkunde*. 1913. Sept. 25. Vol. 31. No. 1. pp. 37-38.

The author wishes to correct what he considers misinterpretations of his original statements regarding *Herpetomonas muscae domesticae* made in 1904. When describing the organism as biflagellate, he had intended to convey that the greater number of the vegetative, large forms possessed two thick rhizoplasts, and two flagella. In division near the two original mother-rhizoplasts two daughter-rhizoplasts grew and from these the two long daughter flagella. Daughter products could be (1) forms with one long and one short flagellum, (2) forms with equal flagella, (3) uniflagellate forms that would acquire a second flagellum later on. WENYON considers the uniflagellate form as the type, and what Prowazek considers a two-division stage, to WENYON would be a four-division stage. By statistical methods Prowazek has found that the greater number of flagellates examined in Rovigno are biflagellate. Small, uniflagellate forms, he considers, are about to encyst, and from his statistics believes the biflagellate form to be the type. That the two workers were dealing with the same organism seems to be accepted by them as apparently they have interchanged preparations.

A. P.

O'FARRELL (W. R.). **Preliminary Note on a New Flagellate *Crithidia hyalommae*, sp. nov., found in the Tick *Hyalomma aegyptium* (Linnaeus, 1758).**—*Jl. Trop. Med. & Hyg.* 1913. Aug. 15. Vol. 16. No. 16. pp. 245-246. With 8 text-figs.

*Crithidia hyalommae* n. sp. occurs in the common cattle tick of the Anglo-Egyptian Sudan, *Hyalomma aegyptium*. The

present note deals with the flagellate stage of the organism, which, together with pre-flagellate and post-flagellate stages, is found in the coelomic fluid of the tick. The flagellates are long and slender, the total length varying from  $26\ \mu$  to  $48\ \mu$ , the flagellum being about  $12.5\ \mu$ . The average breadth is  $2.5\ \mu$ . The posterior end tapers to a point. There is a well marked undulating membrane. The nucleus is roundish or oval; in quiescent stages a well defined central karyosome is present, but when active division or cyst formation is about to occur, the nucleus becomes granular. The blepharoplast is usually on the flagellar side of the nucleus. Division is initiated by that of the blepharoplast, one of the daughter blepharoplasts moving to the aflagellar aspect of the nucleus, which thus lies between the two blepharoplasts. The flagellum arises near the blepharoplast but is not connected with it.

The organism usually moves with the flagellar end forwardly directed, but in turning movements and for short distances the aflagellar end can be forwardly directed. The mechanism of movement is briefly described.

Five figures of the ordinary form and three of the dividing flagellate are given. The full paper will be awaited with interest.

A. P.

FRANCHINI (G.). *Nuovo Contributo allo Studio dell' Haemocystozoon brasiliense*.—*Ann. di Med. Navale. e Colon.* 1913. May-June. Anno 19. Vol. 1. No. 5/6. pp. 477-480. With 5 text-figs.

The present paper of Franchini covers practically the same ground as his paper published in the *Bulletin de la Société de Pathologie Exotique* of May, 1913, which has already been reviewed with illustrations. [See this *Bulletin*, Vol. 2, pp. 265-266.] All his previous findings are confirmed and he emphasises that further search has revealed no form of leishmania, either free or enclosed in leucocytes, and because of its different development from herpetomonad parasites, as it encysts in the peripheral blood, he retains the name *Haemocystozoon brasiliense*.

A. P.

MOLDOVAN (J.). *Beitrag zur Entwicklung des Leucocytozoon Ziemanni (Laveran)*. [Development of *L. Ziemanni*.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Sept. 27. Vol. 71. No. 1. pp. 66-69.

The author describes the schizonts of *Leucocytozoon ziemanni* in two screechows. The infection with the protozoon was spontaneous. Typical gametes occurred in the blood. Smears of the lungs, heart, brain, liver, kidney and bone marrow contained endocellular schizonts of various sizes and in different stages of nuclear division, as many as 30 nuclei being found. The spleen was not examined. The young schizonts show a central nucleus with peripheral chromatin masses, the cytoplasm being alveolar. With growth and nuclear division large ovoid schizonts

are produced, with as many as 30 tiny nuclei. Following the nuclear divisions, the cytoplasm commences to segregate around the nuclei, and these become separated into groups and then probably into uninucleate forms giving rise to merozoites. The last two stages have, however, not been observed. The author notes the resemblance of his schizonts to those described by FANTHAM for *Leucocytozoon lovati* but considers there are some differences. [Full merozoite formation and liberation has been observed in *L. lovati*.]

The author describes also the young gametes, which are of the usual type. The figures show six successive stages in the growth and nuclear multiplication of the schizont.

A. P.

PHISALIX (Marie). *Essai d'Infection sur la Vipère aspic et les Couleuvres Tropicodonotes avec Haemogregarina roulei*.—*Compt. Rend. Soc. Biol.* 1913. July 25. Vol. 75. No. 27. pp. 110-111.

Hitherto it has been considered that *Haemogregarina* infections are not transmissible by inoculation. Mme. Phisalix has tried to transmit *Haemogregarina roulei*, parasitic in *Lachesis alternatus*, to other snakes, using *Vipera aspis*, *Tropicodonotus natrix* and *T. viperinus*, known to be uninfected.

1.5 cc. of liver pulp or blood of *Lachesis alternatus*, containing both free and endoglobular forms of the haemogregarine, were deposited direct on the pulmonary epithelium or the gastric mucosa of the snakes. The liquid was passed into the trachea drop by drop from a syringe with a soft canula, and a similar process was used in depositing the liquid on the gastric mucosa. Sixteen snakes, in four groups of two adders and two vipers each, were thus inoculated; eight with liver pulp, eight with blood. In one only, a viper that received 1.55 cc. of blood in its lung and that died 25 hours after inoculation, were haemogregarines found, these being represented by a few free and endoglobular forms in the heart blood. The lung still contained a little of the injected blood with living parasites in it. In *Lachesis* the free haemogregarines were about five to every hundred erythrocytes, and therefore would be very scarce in the blood of the inoculated snake, but no multiplication in the blood seemed to have occurred. New infection is thought to depend on the receptivity of the subject, rather than on the nature of the haemogregarine introduced.

A. P.

PHISALIX (Mme.). *Formes de Multiplication d'Haemogregarina roulei chez Lachesis alternatus*.—*Compt. Rend. Soc. Biol.* 1913. Aug. 7. Vol. 75. No. 28. pp. 194-196.

Mme. Phisalix has found two forms of cysts of *Haemogregarina roulei* in the liver, lung, spleen and kidney of the viper, *Lachesis alternatus*, the free and endoglobular forms of the parasite having been described in a previous paper. (See this *Bulletin*, Vol. 2, p. 263.)

Cysts with macromerozoites were found in smears of the above organs and sections of the same showed them localised in capillaries. The cysts are regular, elliptical, thick walled, containing one to six nuclei according to the stage of division. They may reach  $35\ \mu$  long and  $25\ \mu$  wide. These merozoites measure  $15\ \mu$  to  $16\ \mu$  long like the young adult forms.

Cysts containing micromerozoites co-exist with those producing macromerozoites. They can reach  $45\ \mu$  long and  $30\ \mu$  broad, are always ovoid, are thin walled, and ultimately contain numerous nuclei. Protoplasm condenses around each nucleus and a large number of small, curved merozoites,  $7\ \mu$  to  $7.5\ \mu$  long by  $3\ \mu$  broad, are set free into the blood stream, where they invade the erythrocytes and develop into adult Haemogregarines.

A. P.

NICOLLE (Ch.) & CONOR (Marthe). La Toxoplasmose du Gondi. **Maladie Naturelle, Maladie Expérimentale.**—*Arch. Inst. Pasteur Tunis*. 1913. No. 1-2. pp. 106-115.

The authors describe first the natural and then the artificial infection of the gondi with toxoplasma. (See this *Bulletin*, Vol. 2, pp. 52-54 and 259-263.) The natural infections were obtained in South Tunisia, whence the disease was first reported. Two kinds of ectoparasites occurred on these naturally infected gondi, an undetermined Ixodus and the larva of a Trombidium. During the first two months half the animals perished, but no toxoplasma were found. Mortality ceased in July, and recommenced at the end of September. From then to January nine animals died, eight with toxoplasma. No special symptom appeared prior to death. In smears toxoplasma were abundant, the spleen containing enormous numbers, the liver and lungs being heavily infected, the blood and bone marrow with few parasites. Polymorphism occurs. Longitudinal division is the only means of multiplication observed. Masses of parasites have been seen but are not considered as identical with the cysts of SPLENDORE, no anterior stages having been seen.

Two cases of experimental infection with bone marrow are described. Incubation takes from eight to nine days. The spleen contains huge numbers of parasites; pseudocysts may be present. Liver and lungs have numerous parasites; kidneys, fairly numerous; peritoneal fluid, very numerous.

Mice are very sensitive to *Toxoplasma gondii*, the parasites being most numerous in the peritoneal fluid and rare elsewhere. Intramuscular injection was used. Guinea-pigs and pigeons also can be infected. Ten white rats, a very young dog, two *Macacus*, two toads and two frogs were refractory.

Protocols of the various animals investigated are given in

A. P.

FANTHAM (H. B.). *Sarcocystis colii* n. sp., a Sarcosporidian occurring in the red-faced African Mouse Bird, *Colius Erythromelon*.—*Proceedings Cambridge Philosoph. Soc.* 1913. July. Vol. 17. Pt. 3. pp. 221-224. With 1 plate.

The paper contains an account of a new species of Sarcosporidia, *Sarcocystis colii*, occurring in the red-faced African mouse bird. This bird is considered by Kaffirs as good eating, and it is possible that the parasites thus reach man. The Sarcosporidian trophozoites (Miescher's tubes) are distributed throughout the skeletal musculature, the large breast muscles being the most heavily parasitised part. They occur in the heart muscle, and scattered in the pericardium, peritoneum and intestinal mesentery. Large Miescher's tubes are 2.5 mm. long and nearly 1 mm. broad. Tubes in the cardiac muscle are smaller than those in the general musculature. The spores (Rainey's corpuscles) are not so large as those of some other Sarcosporidia, being only about  $7\mu$  long. They exhibit polymorphism, two main types being distinguished, namely, narrow, sickle-shaped bodies with more deeply staining contents, and broader spores with paler contents, which sometimes are seen in process of division. The nucleus shows cyclical structure, sometimes appearing as vesicular with a karyosome, at other times the chromatin showing as evenly distributed granules. The nucleus is usually near the blunter end, and a polar vesicle is often seen near the more pointed end. The spores show no marked metachromatic granules. Portions of probable polar filaments have been seen in a few well-stained specimens. The characters of the new species, *S. colii*, are polymorphism and somewhat small size of the spores, the apparent lack of marked metachromatic granules and the presence of a definite polar vesicle. The paper is illustrated with a photograph of the dorsal aspect of *Colius erythromelon*, approximately natural size, showing the distribution of the Miescher's tubes.

A. P.

KNOWLES (R.) & ACTON (Hugh W.). A Note on Kurloff Bodies.—*Indian Jl. Med. Research.* 1913. July. Vol. 1. No. 1. pp. 206-211. With 2 plates.

The paper first gives a brief summary of the current opinions as well as past ideas regarding the nature of the Kurloff bodies found in the blood of guinea-pigs. Thirteen out of eighteen healthy adult guinea-pigs contained Kurloff bodies, and four out of six young ones. Forty films from a guinea-pig showing numerous Kurloff bodies were made at the same sitting and batches of eight films each were:—“(1) allowed to dry in air and then fixed with methyl alcohol; (2) fixed whilst wet in methyl alcohol; (3) fixed while wet with formol-alcohol (formalin 1: absolute alcohol 9); (4) fixed whilst wet with Schaudinn's granule fixative (saturated aqueous perchloride 60 cc., absolute alcohol 30 cc., glacial acetic acid 3 cc.); (5) fixed whilst wet by immediately plunging them into Schaudinn's fixative, kept at 60° C. on a water bath (saturated solution of perchloride in saline 2 parts: absolute alcohol, 1 part).” Of each group, two films were

stained by Leishman, two by Giemsa, two by original Romanowsky, two by Mallory's iron-haematoxylin counterstained eosin. The result showed that the appearance of Kurloff bodies in fixed films depends almost entirely on the fixative.

The Kurloff bodies were also examined by the jelly method by dark ground illumination and by the dahlia solution method.

The authors' conclusions are as follows:—

"(1) No reliance can be placed upon the appearance of the Kurloff bodies as seen in fixed films, unless the action of the fixative fluids has been previously investigated in order to see what fixation images are obtained. Otherwise the different fixation images obtained may be readily mistaken for stages in a cycle of development.

"(2) In Kurloff bodies stained by a dahlia solution and examined on a warm stage we have been unable to trace any cycle of development.

"(3) It seems somewhat unlikely—though not impossible—that a structure seen in the blood of 71 per cent. of apparently perfectly healthy guinea-pigs is parasitic in origin.

"(4) We adhere to the view that they are cell inclusions of a non-parasitic nature.

"(5) We believe that the Kurloff body is a vesicular structure, and probably of cytoplasmic origin. The guinea-pig is an animal in which these cytoplasmic structures are particularly well developed, *e.g.*, the large archoplasmic vesicles in the testicular cells.

"(6) In the course of repeated examinations of the blood of pigeons in connection with a study of the Halteridium parasite we have occasionally seen somewhat similar bodies in the large mononuclear leucocytes and we doubt if the Kurloff body is specific to the guinea-pig."

A. P.

DA ROCHA-LIMA (H.). Zur Demonstration über Chlamydozoen.—*Verhandlungen der Deutschen Pathologischen Gesellschaft.* 1913. pp. 198-210. With 10 text-figs.

The paper contains an account of the Chlamydozoa in relation to disease. The author states care must be taken to differentiate between Chlamydozoa and structures having a similarity to them. By histological methods the Chlamydozoa-inclusions appear granular, like microsomes. They are not artefacts produced by the fixatives employed, since they can be seen in the fresh state, *e.g.* in *Molluscum contagiosum*. In other cases they are more difficult of observation. The elementary bodies occur in the virulent filtrate after use of a bacteriological filter, but are lacking in the non-virulent filtrate from a colloid filter. The elementary bodies thus appear as the excitants of disease. Another hypothesis considers them as degeneration or reaction products of the cell. This idea also is discussed.

The initial bodies that give rise to the elementary bodies in variola-vaccine form a plastin-like coat for themselves. The initial body (Guarnieri corpuscle or body) usually appears homogeneous.

The role of Chlamydozoa in various forms of cancer is discussed in some detail, and the author pleads for fuller investigations of the organisms, of which he could only give a brief outline. Ten figures illustrate the appearance of various Chlamydozoa within their host cells.

A. P.

BREINL (Anton). Protozoa encountered in the Blood of Native Australian Animals.—*Australian Institute of Trop. Med. Report for the Year 1911.* pp. 30-38.

The report contains notes on trypanosomes, plasmodia, proteosoma, haemogregarines and Haemoproteus from native Australian animals.

Trypanosomes.—*Trypanosoma lewisi* was present in 15 per cent. of the rats. *T. majus* occurred in the white headed sea eagle, *Haliastur girrenera*, and *T. avium* in *Falco hypoleucus*, in whose heart blood rosettes of spindle shaped parasites, suggesting cultural forms, were found. Bower birds, *Chlamydodera orientalis*, harbour a new trypanosome, *T. chlamydoderae*. It is dimorphic and differs from *T. avium* in size and in its small nucleus and blepharoplast. *T. notophoyx*, n. sp. occurs in the blood of the blue crane, *Notophox novaehollandiae*. *T. chelodina* occurred in the tortoise, *Chelodina longicollis* and a trypanosome was found in the owl, *Ninox boobook*.

*Proteosoma praecox* occurred in *Falco hypoleucus*.

*Plasmodium pteropi*, n. sp. was present in the flying fox, *Pteropus gouldi*.

Haemogregarines are described from *Chelodina longicollis*. They have been found in numbers in the guts of the tick *Aponomma trimaculatum*, infesting *Varanus varius*, parasitised by *H. varanicola*. The snakes, *Dipsadomorphus fuscus* and *Python spilotes* var. *variegatus*, also harbour Haemogregarines. A leucocytozoon was found in the friar bird, *Tropidorhynchus corniculatus*, and also *Haemoproteus danilewski*. *Haemoproteus noctuae* was present in *Ninox boobook*, *H. columbae* in the pigeon, *Ptilopus superbus*, and *H. danilewski* in birds of several species.

A. P.

LEGER (Marcel). Hématozoaires d'Oiseaux de la Corse.—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 515-523.

The author examined the blood of various birds obtained in the Eastern part of Corsica in the province of Casabianda, and found various trypanosomes, Haemoproteus, Leucocytozoa, and a Microfilaria. Trypanosomes occurred in *Fringilla petronia* and *Caprimulgus europaeus*. The trypanosome from the *Fringilla* is identified as *T. laverani*, NOVY and MACNEAL. A new species, *T. thiersi* is created for the trypanosome from the *Caprimulgus*, mainly on account of its great size, 84.75  $\mu$  long, 8.75  $\mu$  broad—the largest of the avian trypanosomes.

Haemoproteus was fairly common in the *Fringilla* and a slight infection occurred in *Corvus corone*.

Leucocytozoa were found in *Fringilla petronia*, *Turtur auritus* and *Corvus corone*. According to Léger, the Leucocytozoon of *Fringilla* is unlike that of other Fringillidae and he creates a new species, *L. gentili*, for it. The Leucocytozoon of *Turtur auritus* is the same as *L. marchouri* of *T. humilis*. The Leucocytozoon of *Corvus corone* is made a new species, *L. zuccarelli*, because (1) it presents peculiar, deepstaining granulations, which



suggest but are not, pigment, in overstained Giemsa smears; and (2) the host cell undergoes nuclear karyolysis even though very young parasites may be present.

A. P.

KÉRANDEL (J.). Trypanosomes et Leucocytozoön observés chez des Oiseaux du Congo.—*Ann. Inst. Pasteur*. 1913. June 25. Vol. 27. No. 6. pp. 421-439. With 2 plates.

The paper deals with trypanosomes and Leucocytozoa observed in birds killed during the expedition of LENFANT in the regions of Haute-Sangha and Haute-Logone, Congo. The author has created several new species of these genera, holding that the same trypanosome or Leucocytozoon only occurs in one specific host bird or in a host very nearly related thereto.

He gives dimensions and description of the trypanosome of the guinea-fowl, *Numida meleagris*, which is probably the same as that described by WENYON from *Numida ptilorhyncha*.

The trypanosome from the francolin, *Francolinus bicalcaratus* is described as a new species, *T. francolini*. It is spindle-shaped with vacuolated protoplasm. There is a much folded, undulating membrane. A distance of 18.9  $\mu$  intervenes between the nucleus and the blepharoplast.

*T. pycnonoti* n. sp. from *Pycnonotus tricolor* is a very rare trypanosome. It was examined fresh and in stained preparations. Its total body length is 49.5  $\mu$  and breadth 5.4  $\mu$ .

*T. viduae* n. sp. from *Vidua serena* is rare. Three specimens were found in a single smear. Its posterior extremity is drawn out to a fine point. No free part of the flagellum is present.

The trypanosome of *Strix flammea trimaculata* was studied in only one well stained specimen. It resembles *T. avium*.

*T. eurystomi* n. sp. was found in the roller, *Eurystomus gularis*. It has a thick body and a regularly folded undulating membrane. Three specimens only were seen.

*Caprimulgus fossei* harbours a large and a smaller trypanosome, described by the author as two new species, *T. caprimulgi majus* and *T. caprimulgi minus*. Detailed dimensions are given. A trypanosome is also described from an unknown bird.

Leucocytozoa were found in most of the birds that also contained trypanosomes. The host cells are considered to be mononuclear leucocytes and erythroblasts. Following the same plan as with trypanosomes, the author creates new species of Leucocytozoa for new hosts. He attaches importance to the degree of deformation produced in the host cell. His Leucocytozoa include:

1. A Leucocytozoön like *L. neavei* from *Numida meleagris*.
2. *L. francolini* n. sp. from *Francolinus bicalcaratus*.
3. A Leucocytozoön from *Strix flammea trimaculata*.
4. *L. caprimulgi* n. sp. from *Caprimulgus fossei*.
5. *L. eurystomi* n. sp. from *Eurystomus gularis*.

Most of the parasites dealt with in the paper are illustrated, one or two figures of each being given.

[Considering that nothing is stated about the mode of transmission of these parasites and that therefore the cycles of the

trypanosomes in the invertebrate hosts are unknown, it does not seem very wise to create new species in several of these cases, especially when small numbers, *e.g.* three individuals, only have been observed.]

A. P.

VON PROWAZEK (S.). Zur Parasitologie von Westafrika.—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. July 29. Vol. 70. No. 1/2. pp. 32-36. With 1 coloured plate.

The author has received blood smears from animals killed by the expedition to Lake Chad, on which further papers are promised. The present paper deals with (i) the structures known as Todd's bodies from the blood of a chameleon. A full account of previous work on the subject is given. The structures were found always associated with other signs of degeneration in the blood. Prowazek describes their appearance and concludes that they are not parasitic since no differentiation between chromatin and cytoplasm can be discerned, nor is there evidence of multiplication. Eight figures are given.

ii. In the erythrocytes of the yellow mouse he has found bodies which he considers allied to the *Grahamella* of the mole. Two figures are given.

iii. Certain smears from various antelopes and gazelles showed gut as well as blood parasites—probably the result of gunshot contamination. Among these is an organism somewhat like *Spirillum sputigenum*, for which Prowazek suggests the name *Selenomonas*. The organism is bowed or sausage shaped, and from its inner concavity a number of united cilia or flagella form a pseudo-membrane. The protoplasm contains alveoli. The outer membrane can become thickened and cyst-like, when the ciliated membrane contracts and resting stages are produced. Nine figures of *Selenomonas* are given.

A. P.

ALEXEIEFF (A.). Introduction à la Révision de la Famille *Herpetomonadidae* (= *Trypanosomidae* Doflein 1911).—*Arch. f. Protistenkunde*. 1913. May 27. Vol. 29. No. 3. pp. 313-341. With 3 text-figs.

The present paper continues the ideas set forth in two previous ones (Reviewed in this *Bulletin*, Vol. 1, pp. 158 & 496). Alexeieff revises the *Herpetomonadidae*, preferring this name to *Trypanosomidae* of DOFLEIN, *Herpetomonas* being a more primitive type of flagellate than *Trypanosoma*. The *Herpetomonadidae* include three genera only, *Herpetomonas*, *Crithidia*, and *Trypanosoma*. Each genus is defined and discussed. As before, the author protests against the idea of absolute specificity of parasites to the one host, and insists that the sole criterion should be the morphological one. The existence of erratic parasites on the one hand, and the great plasticity of living organisms on the other both need full consideration.

The genus *Herpetomonas* is considered to have three good species. I. *H. muscae domesticae* (= *Leptomonas drosophilae*,

= *L. pycnosomae*, = *L. muscae domesticae*, = *H. calliphorae*, = *L. ampelophilae* = *H. luciliae*). II. *Herpetomonas gracilis* (= *H. sarcophagae*, = *Cercoplasma (Leptomonas) mirabilis* = *C. (L.) mesnili*, = *C. caulleryi*). III. *Herpetomonas jaculum* (= *L. jaculum*, = *H. lygaei*, = *L. agilis*, = *L. davidi*).

The genus *Crithidia* is discussed in some detail, is considered autonomous, and is defined as having the blepharoplast in a juxta-nuclear position in the flagellate form. The author does not think the genus *Rhynchoidomonas* then is necessary, and that forms intermediate to *Crithidia* and *Rhynchoidomonas* will be found.

Trypanosomes are not considered to be specific to one host only. There are more than 40 species of Trypanosomes recorded from mammals alone. The blood of mammals has a constant and uniform constitution, and the contained trypanosomes are practically morphologically identical. Physiological criteria such as inoculability, acquisition of immunity and pathogenic action, all are variable, and thus cannot be used for determining specificity. Many "species" should be more accurately described as "races" of a species.

The discussion of heterogamy and autogamy leads to the conclusion that nothing positive is known of sexuality in the Herpetomonadidae, but sexual processes may be replaced by some caryoplasmic relationship.

A. P.

POCHE (Franz). *Das System der Protozoa.* — *Arch. f. Protistenkunde.* 1913. Sept. 12. Vol. 30. No. 3. pp. 125-321. With 1 text-fig.

The classification of the Protozoa is set forth by Poche in 196 pages of text. The phylum Protozoa is subdivided into a complicated and somewhat confusing series of superclasses, classes, under classes, orders, supersuborders, suborders, supertribes, tribes, subtribes, families and subfamilies. More than 200 new names distributed between the above subdivisions appear. Unfortunately the characteristics of the various members as above indicated are often not clearly defined, and though consistency in classification is aimed at, yet gaps occur as can only be expected. Uniformity in the terminology used in classification appears to be the ideal of the author, but the rather inelastic system proposed by him does not admit of progress of knowledge regarding many organisms, and it is easy to see that confusion would be bound to occur when organisms considered first as "families" were, perhaps, found to be "genera" of some other "family" or "order," or when the elevation of a "tribe" to an "order" became necessary with increased knowledge.

The paper does not appear to be of much interest to the practical worker in the tropics.

A. P.

## REVIEWS.

MENSE (Carl). *Handbuch der Tropenkrankheiten. Zweite Auflage. Band I. Die Krankheitserreger und Krankheitsüberträger unter den Arthropoden.* [Disease-Producing and Disease-Carrying Arthropods.] Von Dr. Adolf EYSEL, R. DOERE und V. RUSS.—xv + 295 pp. With 200 text-figs., 10 black and white and 2 coloured plates. 1913. Leipzig: Verlag von J. A. Barth. [Paper cover, 16.20 Mk.: bound, 18 Mk.]

This book gives a plain unvarnished account—not quite up to date, perhaps, in some particulars—of all the Arthropoda that are of acknowledged importance in human pathology—both those that are constantly concerned in the transport of pathogenic organisms, and those less direful forms that are only occasionally noxious as temporary parasites or as inflicting irritating or venomous wounds.

The disease-carriers included are—besides *Cyclops*, which is disposed of in a few lines and a figure—ticks, lice, bugs, fleas, mosquitoes and other midges, gadflies, Muscidae, and tick-flies; every group being separately considered in its general characters, anatomy, mode of life, habits, classification, methods of preservation, and prophylaxis. Natural enemies are sedulously enumerated in every case; but the author does not consider the broad question whether they are to be accepted as of supreme use to the sanitarian under all circumstances, as some are inclined to imagine, or whether—as seems more reasonable to suppose—they are to be regarded merely as humble auxiliaries in places where they themselves are protected and assisted by those artificial conditions which the sanitarian maintains in opposition to the scheme of nature. To every group, too, an extensive bibliography is appended, which will be of the greatest service to medical men who have to grapple with the sanitary problems of tropical countries.

Though the *Culicidae* occupy nearly one-third of the volume, not everything that might with advantage be said about them is included. Their structure and life-history, their method of feeding, the mechanism of the emergence of the imago from the pupal skin, and the best ways of collecting and preserving them are described very fully, while their bibliography runs to 38½ pages. But, on the other hand, not very much is said about approved methods of destroying larvae, and all *Culicidae* except the Anophelines are deliberately ignored—a few lines and a couple of figures being all that is accorded even to *Stegomyia*; here the author carries to an unreasonable extreme his opinion that the medical man should be content with a limited knowledge of the group, and perhaps hardly justifies his exhaustive bibliography.

One may sympathise with the author's impatience of cabinet-entomologists whose "genera" and "subfamilies" of *Culicidae* sometimes dissociate—what no man should sunder—the male and female of the same species, and whose "Anopheline genera" sometimes merely magnify individual or seasonal variations; and many will join with him in qualifying with inverted commas divisions so arbitrary and unnatural. But it is hardly satisfactory to find all the species of *Anopheles* left on the cabinet-entomologist's shelf, on the ground that *all*—whatever for the moment be the verdict of the laboratory—may possibly, in some still undetermined circumstances, harbour malarial parasites.

The author tabulates the differences between *Anopheles* and all other *Culicidae* very clearly, but whether these differences justify the isolation of the former in a separate family, *Anophelidae*, is a very doubtful question. This exaggeration of differences, in disregard of obvious fundamental resemblances, is the very fault that the author condemns with so much justice in the troublesome *Fachentomolog*: a natural classification must take likenesses into account equally with unlikenesses, and must represent the sum of both. The author, probably, would be the first to admit that it is not a matter of indifference, from a practical standpoint, whether a classification be natural or arbitrary; for, as HUXLEY insisted, a natural classification is of perennial importance since it represents a series of accurate generalisations regarding structure.

In the section on the *Muscidae*, the common house-fly hardly receives the attention it deserves; there may be a tendency in some quarters to aggrandize the pathogenic importance of this insect, but every medical man in the tropics ought to know all that is to be known about it.

The systematic account of *Glossina* does not go beyond AUSTEN'S "Monograph" of 1903; the relation of *G. morsitans* to human trypanosomiasis is not referred to; and though animals that might prove to be natural enemies of tsetse-flies are enumerated, the far more important subject of animals that may assist tsetse-flies in their evil work—and how these are to be regarded by the sanitarian—is not discussed.

Of other blood-sucking Muscids *Stomoxys calcitrans* is the only species that receives much attention. *Beccarimya* makes an unfortunate re-appearance, and *Glosinella* again emerges from oblivion.

The Arthropoda that are occasionally and individually noxious naturally occupy only a small part of the book. They include Linguatulida, Mites, the larvae of bot-flies, and the larvae of those several families of *Muscidae* that make up the tale of myiasis; all these are considered in detail. Spiders, Scorpions, Centipedes, Aculeate Hymenoptera, certain beetles, and certain Lepidopterous larvae are also briefly dealt with, chiefly in respect of the injuries they inflict.

An appendix on the genus *Phlebotomus* is contributed by DOERR and RUSS; it gives all that is known about the structure, development, habits, life-history etc. of these midges, and all the species, except those quite recently described, are mentioned.

On the whole the volume can be recommended to medical men employed in the tropics as a most laborious and methodical epitome of the Arthropoda that immediately concern them; while to those who have to take up any specific investigation, the bibliographical sections, apart from anything else, will be of signal service.

A. Alcock.

PROUT (W. T.). *Lessons on Elementary Hygiene and Sanitation, with Special Reference to the Tropics.*—xx. + 184 pp. Illustrated. 1913. London: J. & A. Churchill. [2s. 6d. net.]

The "lessons" have now reached a third edition, this testifying to their popularity and general use. Before their first publication there was a special want for a book of the kind and it must be gratifying to the author to find that his work has so successfully filled this void. There is no question of the value of such instruction in schools and amongst the general laity in the Tropics. It is by means such as these that the coming generations will enjoy improved health and all the benefits and advantages that such a state of affairs implies. Dr. Prout's little book is an excellent one.

G. C. L.

## TROPICAL DISEASES BUREAU.

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## PELLAGRA.

SILER (Joseph F.) & GARRISON (Philip E.). *An Intensive Study of the Epidemiology of Pellagra. Report of Progress.—Amer. Jl. of the Med. Sciences.* 1913. July. Vol. 146. No. 1. (No. 496.) pp. 42-66; and Aug. No. 2. (No. 497.) pp. 238-277.

This is a preliminary report of the epidemiological work of the Thompson-McFadden Pellagra Commission during 1912, which was continued in 1913. Spartanburg County in South Carolina was selected for intensive study within a limited area. The Commission studied 282 cases between June and October and found three females to every male attacked. It is believed that household association, which may or may not include family relationship, is an important factor in the distribution of pellagra.

There is the history of a woman who died apparently from pellagra in 1894, but the disease was not recognized in the county until 1909. During 1912 there were 376 cases, including those which had been returned in former years; of this number 12 per cent. died. It is to be noted that 5 per cent. died during the initial attack contracted in 1912. It is suspected that pregnancy inhibits the development of pellagrous symptoms, but on the other hand the list of recent illnesses "possibly predisposing to pellagra" is headed by the entry "general poor health (usually following childbirth)." [An apparent improvement during pregnancy followed by deterioration after childbirth reminds one of certain cases of phthisis.] Only one case of uncinariasis was encountered.

The report includes several diagrams and photographs of wells and of typical houses of patients, all vastly superior to the dwellings of Italian and Egyptian peasants. But these Spartanburg "houses are practically infested with *Cimer*, and the grooves between the boards used in sealing the rooms afford an ideal place for the hatching out of broods of these insects." The domestic fly was present in all and *Stomoxys calcitrans* very often.

Bacon (cured pork) is eaten "either daily or habitually by 92 per cent. of the mill-village group, 87 p.c. of the rural group, and 76 p.c. of the urban group." "We are quite certain, from

personal observation, that the average dietary of the poorer classes of the population in the county studied is much superior both in its variety and actual nutritive value to the dietary of the peasants in the North of Italy." In 83 per cent. of the cases examined economic conditions were bad, and pellagra was found to be most prevalent among people of insufficient means.

Investigation failed to discover any epidemiological evidence pointing to maize as an etiological factor and two cases were met with which denied all maize consumption for two years before the onset of symptoms.

F. M. Sandwith.

LAVINDER (C. H.). Pellagra. Prevalence and Geographic Distribution in Arkansas, Oklahoma, and Texas. — *U.S. Public Health Rep.* 1913. July 25. Vol. 28. No. 30. pp. 1555-1558.

This is an official postscript to the article published on December 13, 1912, relating to the prevalence of pellagra in the United States (see this *Bulletin*, Vol. 1, p. 299). 4,045 cases are now added, of which more than two-thirds occurred in Texas. These figures bring the total number recorded between 1907 and 1912 to 19,915, with the enormous death-rate of 40.59 per hundred reported cases. In the State of Oklahoma this calculated death-rate reached the maximum of 54.91; most of these reported cases were in white people. These three States lie somewhat on the border of what was thought to be the intensive pellagra area. "This suggests rather strongly that pellagra is increasing the area in which it is present and invading new territory."

F. M. S.

BEALL (K. H.). Pellagra.—*Texas State Jl. of Med.* 1913. Aug. Vol. 9. No. 4. pp. 129-131.

As an instance of the danger which pellagra is to a virgin country the author states that this disease in the one State of Alabama killed 446 people in the year 1912, besides invaliding several thousand others. The incidence among children in Texas is comparatively slight, while the sex incidence is similar to other parts of the United States, from three to five women affected to every male adult. The author asks why American women are attacked in greater proportion than European women. He has known a number of cases (three within a week) in which the first definite sign of pellagra developed several days after a surgical operation. As pellagrins are poor subjects for surgery, he wonders whether the disease could be diagnosed earlier by activating the latent skin eruption by means of light, heat or chemical irritation.

In the discussion which followed the reading of this paper, H. L. MOORE (Dallas) stated that the disease had been more or less prevalent for 15 or 20 years in a large orphan home; for the last five years it had been diagnosed as pellagra; before that time it used to be called "Red Disease."

F. M. S.

**SHROPSHIRE (Walter).** *Pellagra as a Public Health Problem.*—*Texas State Jl. of Med.* 1913. Aug. Vol. 9. No. 4. pp. 131-133.

The author considers pellagra to be even more dangerous than tuberculosis, because the American has developed some resistive power to the latter; also we know something about its cause and prevention. Since pellagra was discovered in Texas about six years ago, there have been more than 400 official deaths from it, besides many others unreported. The writer considers that it bears a very striking resemblance to trypanosomiasis and he "cannot see how we could for a moment entertain the proposition that maize produces pellagra."

F. M. S.

**GRIMM (R. M.).** *Pellagra: Some Facts in its Epidemiology.*—*Jl. Amer. Med. Assoc.* 1913. May 10. Vol. 60. No. 19. pp. 1423-1427.

This is a condensed report upon field work in 1911-12 in South Carolina, Georgia and Kentucky, which has already been reviewed [see this *Bulletin*, Vol. 1, pp. 686-688.]

F. M. S.

**ALESSANDRINI (Giulio), GIANNELLI (A.), & FILENI (E.).** *La Pellagra nella Provincia di Roma.*—*Poluclico.* Sez. prat. 1913. Aug. 24. Vol. 20. No. 34. pp. 1213-1221.

The author and his two colleagues were commissioned in the summer of 1912 to examine pellagra patients in the province of Rome and to study the questions which, directly or indirectly, influence the development of the disease. They found that many people returned as pellagrins did not show the least appearance of the disease. Excepting two endemic foci, one in the north and one in the south, they did not find many pellagrins in the province.

Women were chiefly affected, 35 among a list of 46 certified and doubtful cases. No confirmation of the Simulium theory was discovered. The peasants live chiefly but not exclusively on maize and one typical case of pellagra was met with in a man in easy circumstances who ate good food, but not maize. The authors found no evidence to incriminate maize, but they think that water may have something to do with the cause of the disease. In the endemic foci they found some peasants suffering from pellagra who habitually drank doubtful water, but the province as a whole is supplied with an abundance of water, furnished by many springs.

F. M. S.

**KLEIMINGER.** *Neue Beiträge zur Pellagralehre.* [New Contribution to the Study of Pellagra.]—*Zeitschr. f. d. gesamte Neurolog. u. Psychiat.* Orig. 1913. May 31. Vol. 16. No. 5. pp. 586-668.

The author writes from a provincial lunatic asylum in Germany and gives a detailed historical account of the disease followed



by a list of predisposing causes, including heredity. He devotes five pages to the various theories of etiology from STRAMBIO to SAMBON. He describes nine of his own cases imported into his asylum, including autopsies of the fatal cases. Most of his patients suffered from katatonia and melancholia. He draws attention to the fact that pellagrous lunatics do not always exhibit the typical symptoms of pellagra as usually described, though all of his cases suffered from dermatitis and enteritis. He believes that the later symptoms, bodily and mental, are due to infection from the intestines. He is a disbeliever in pellagra *sui generis*; "in default of an uniform etiology, we can only speak of a pellagra-symptom-complex." Metabolism is responsible for the predisposition; the nature of the disease is an infectious intestinal catarrh. The erythema is due to intestinal toxins acted on by outside stimuli. Non-specific nervous disturbances are produced as the result of chronic intoxication. Endemic pellagra can only be combated by raising the status of the poorer inhabitants, so that all may have a sufficiency of animal food. The author recommends that the State shall encourage the breeding of cattle and the initiation of fish ponds, while the police should see that none but healthy maize is consumed.

He considers that the spread of pellagra in Germany is unlikely because the people are all well fed.

F. M. S.

STANNUS (Hugh S.). *Pellagra in Nyasaland*.—*Ann. Med. Rept. on the Health and Sanitary Condition of the Nyasaland Protectorate for the Year ended 31st March, 1913*. pp. 78-86. (London: Printed by Waterlow & Sons, Ltd.).

The author discovered the existence of pellagra in 1910 in the Zomba prison, and during the 12 months ending in March 1913; he has now seen 131 cases among the prisoners, besides others in adjoining villages. He points out the habitual symmetry of the rash, but tells of one patient who sat with his back bared to the sun every day, because he thought it eased his pain, and his whole back became covered with the eruption. He says that some of his cases had to be diagnosed in spite of the complete absence of rash for long periods of time. In such cases he was assisted by the fairly constant presence of non-syphilitic "rhagades" at the angle of the mouth, "consisting of sodden and thickened epithelium with cracks which appear white on the black skin." The affection of the free margin of the prepuce is exactly similar. Epigastric discomfort and pain are very frequent symptoms and one insane pellagrin refused food and had the delusion "that there were flies in his stomach which were biting him." The tongue originally coated, exfoliates, and leaves the tip and sides bright, smooth and shining, devoid of visible papillae. These Nyasaland cases are complicated by ankylostomiasis and bilharziasis of the rectum, but troublesome diarrhoea was also present, apparently due to the pellagra. Cases were seen among the prison warders and native troops, but never among the Indian contingent, numbering 70 Sikhs. Men are more attacked than women. "Men in the Central Prison, Zomba, developed pellagra

who had been under observation in the prison for years, and who had had no maize for years or, in other cases, only in very small amount." The prison ration consists of one and a half pounds of rice daily, with a little salt. The writer draws attention to the well-known habit of pellagrous symptoms to disappear and then to relapse in the following year. He believes that this so-called relapse is possibly a seasonal re-infection or intoxication.

(Conclusions:—

"1. Pellagra is endemic in Nyasaland.

"2. The outbreak in the Central Prison has assumed the proportions of an institutional epidemic.

"3. All maize theories of causation, as such, are disproved.

"4. There is some evidence in favour of the disease being caused by an intoxication due to the ingestion of damaged grain, whether it be rice, maize or other.

"5. The theory of an infection carried by *Simulium* (Sambon) receives very considerable support, and so far in this country there are no facts militating against that theory.

"It is to be carefully noted, however, that the data brought forward equally support the theory which lays the cause of the disease at the door of malnutrition, that is, nutrition wanting in some necessary principle."

[This paper is well worth reading.]

F. M. S.

Box (Charles R.). *Pellagra.—Practitioner*. 1913. June.  
Vol. 90. No. 6. (No. 540.) pp. 940-951.

This paper gives an accurate resumé of the disease, culled from the books and pamphlets of modern writers on pellagra. The author reminds us that maize is not largely used in England as human food. The best known derivative is sold as "corn-flour." Popcorn is torrefied maize and, curiously enough, Dr. Box's two boy patients were fond of it, but they only had the means to buy it occasionally, and it can hardly be suggested that this preference was in any way a cause of the disease.

A maize flour, known as "cones," is sold to bakers, who powder their hands with it when mixing dough—[The best bakers state they use rice flour for dusting their hands and boards]—The author recommends arsenic as the most valuable drug treatment, such as Fowler's solution in doses gradually increased to 20 or 30 minims three times daily.

F. M. S.

#### ETIOLOGY.

SANDWITH (F. M.). Is Pellagra a Disease due to Deficiency of Nutrition?—*Trans. Soc. Trop. Med. & Hyg.* 1913. April.  
Vol. 6. No. 5. pp. 143-148.

The author summarises three prominent theories of the etiology of pellagra.

(1) The maize theory initiated by STRAMBIO in 1786.

(2) The photodynamic theory of RAUBITSCHER, an adaptation of the views of RAPOLLI (1771).

(3) The protozoal theory of SAMBON, advanced in 1905.

Bad food as a cause was first suggested at the end of the eighteenth century. The recent developments in connection with

beriberi have caused the author to wonder anew whether pellagra also is not due to deficiency of nutrition. He indicates the points of resemblance between the two diseases. He points out that since 1885 beriberi has been practically eradicated from the Japanese navy by improving the rations. He himself always found that *early* cases of pellagra in Egypt could be cured by admitting them to hospital and giving them an abundant mixed diet, which was distinctly better than that which they obtained in their own homes. Among the products of protein decomposition is the necessary tryptophane  $C_{11}H_{12}N_2O_2$ . This is present in nearly all proteids, but is shown to be entirely absent from zein, the proteid of maize. He suggests that the inferior maize eaten by peasants in Italy and Egypt "may contain some complex organic body which by virtue of a slight chemical change cannot be used in the normal chain of living processes." He points to the effectiveness of atoxyl, a para salt, and the ineffectiveness of its meta and ortho forms, and to the minute chemical changes which may reduce the efficiency of adrenalin by one half. Ultra violet rays have, he shows, the effect of accelerating most organic chemical reactions. Finally, he refers to the Report of the Pellagra Commission of the State of Illinois, recently published. This Commission concluded that according to the weight of evidence pellagra is a disease due to infection with a living micro-organism of unknown nature, but also stated that deficient animal proteid in the diet may constitute a predisposing factor.

A. G. B.

JENNINGS (Allan H.) & KING (W. V.). **An Intensive Study of Insects as a Possible Etiologic Factor in Pellagra.**—*Amer. JI of the Med. Sciences*. 1913. Sept. Vol. 146. No. 3. (No. 498.) pp. 411-440.

This valuable contribution, emanating from the Bureau of Entomology of the U.S. Department of Agriculture in Washington, is the result of the appointment of the authors, early in 1912, by Dr. L. O. HOWARD, to investigate the possible relation of insects to pellagra. The report deals chiefly with field work from June to October, 1912, with the Thompson-McFadden Pellagra Commission in the Spartanburg County of South Carolina. The population of this County is 83,465, and 28 cotton mills supply the chief industry. The pellagra cases were distributed among the population as follows: mill hands 151, rural 77 and urban 51. The proportion of female to male cases was three to one, which is the approximate ratio of pellagra throughout the United States. The authors find "that a close correlation appears to exist between pellagrous incidence and the amount of time spent in or about the home, and that this holds, not only when the sexes are compared, but within the sexes themselves." Comparison is drawn with two other diseases, chiefly of rural nature, malaria and acute anterior poliomyelitis. The bed-bug (*Cimex lectularius*) was found to be almost always present, and was admitted in 241 out of 256 cases. The human flea (*Pulex irritans*), on the other hand was rarely found and there was no

evidence to incriminate fleas derived from cats, dogs and poultry. It is interesting to note that the fleas with which the only rat examined was infested were *Xenopsylla cheopis*. The mosquitoes chiefly met with were *Culex quinquefasciatus* and *C. restuans*. Six pages, including two good illustrations of breeding places, are devoted to the consideration of Simulium and SAMBON'S "attractive theory" and conclude thus: "The facts as set forth above do not support the incrimination of Simulium in the transmission of pellagra."

The authors agree with those who consider that the essential is not to find that an occasional individual insect might be a potential transmitter of disease, but rather that the group lacks the habits of close association with man, necessary to the establishment of the biologic relation between the vertebrate host, the invertebrate host and the parasite of the disease. They maintain that Simulium is essentially "wild" and therefore not more likely to be dangerous to man than forest mosquitoes in malaria. "The assertion that these flies cause severe epizootics is certainly not supported as regards America at least." "Species of Simulium are unquestionably found in numbers in large areas of country in which pellagra is rampant, but there is evidence to show that, contrary to Sambon's assumption, the disease is found where Simulium is not." The authors state, on the authority of the Imperial Entomologist of Barbados, that the physical characteristics of that island entirely preclude the existence there of this fly. It is considered that the ubiquitous house fly must continue to be regarded with suspicion.

But it may be seen from the following Summary, which is too valuable not to be reprinted, that the authors still believe that if any insect is responsible it may be Stomoxys.—

"Our investigation was entered upon with no bias in favour of the infectiousness of pellagra or its transmissibility by insects. In the nature of the case, however, and as a basis for our work, it was necessary to assume that both were among the possibilities.

"No preconceptions as to the involvement of any particular insect were indulged, and our conclusions regarding individual species of insects are based upon the epidemiological picture of pellagra, mainly as presented in Spartanburg County, and upon the habits and distribution of the insects as observed by us and as elsewhere recorded.

"Ticks, lice, bed bugs, cockroaches, horseflies, fleas, mosquitoes, buffalo gnats (*Simulium*), house flies, and stable flies (*Stomoxys*) were under consideration. Of these, horse flies have nothing and cockroaches little to support them.

"Ticks and fleas are excluded on account of their scarcity and the nature of their biting habits. In view of these characteristics, it is doubtful if even the existence of an animal reservoir of infection would bring the groups into prominence.

"Lice and bed bugs do not account for the sex or age incidence or the rural nature of the disease; the scarcity of the former is an additional reason for its exclusion.

"The rarity of mosquitoes here and the lack of coincidence between their distribution and that of pellagra for the State in general, together with the night-biting habits of the local species, which fail to account for the sex incidence, seem sufficient cause for their elimination.

"House flies (*Musca domestica*) should be active if the malady is an intestinal infection in which the germ is passed with the feces, with contaminated food acting as the vehicle of infection.

"The buffalo gnats (*Simulium*) should be eliminated, principally by the facts of their biting habits and lack of those of intimate association with

man, also possibly by their comparatively moderate abundance (in our territory). We find that in Spartanburg County they are hardly known as a pest of man and when they do attack him, it is very locally and largely confined to field workers. Had SAMBON's theory not been advanced, these flies could hardly have attracted suspicion of any connection with pellagra in this country.

"The stable fly (*Stomoxys calcitrans*) displays certain salient characteristics which seem to qualify it for the role of a transmitter of pellagra.

"The range of this one species covers and exceeds that of pellagra; its seasonal activity, likewise, is coincident with that of the disease and, although its period of greatest abundance is somewhat later than the maximum intensity of pellagra, its appearance in spring precedes that of most of the spring recurrences and new cases, at which time it is already abundant; it is an abundant species, its abundance being most manifest in rural districts thus corresponding with the rural nature of pellagra, its numbers amply fulfilling our conception of those necessary to effective disease transmission; it bites by day only, thereby offering an explanation of the phenomenon of sex incidence and the related one of age distribution; it is intimately associated with man and habitually infests his vicinity and enters his dwellings; it bites men frequently and persistently; its longevity seems sufficient for the development of a hypothetical causative organism; it is readily and frequently carried long distances and might thus account for the occurrence of sporadic cases of the disease."

F. M. S.

ROBERTS (Stewart R.). *The Analogies of Pellagra and the Mosquito*.—*Amer. Jl. of the Med. Sciences*. 1913. Aug. Vol. 146. No. 2. (No. 497.) pp. 233-238.

The first words of this paper, written by the Professor of Medicine of Atlanta, are: "with the publication of SAMBON's Progress Report, in 1910, the investigation of pellagra really began." He accepts the theory that pellagra is an infectious, insect-borne disease, but advances several objections against the view that the Simulium is the specific insect carrier. He inclines strongly to the belief that pellagra must be conveyed in some similar way to malaria, yellow fever, filariasis and dengue. He states that pellagra is to Italy and Roumania what yellow fever is to the West Indies, and what malaria is to Greece and to the valleys of the Himalayas. He concludes by asserting that in pellagra "the mosquito accounts for its seasonal relations, periodicity and recurrences; its endemic relations, rural habitat, and sporadic appearances in cities; its peculiar geographical situation in Africa, Europe and America; its predominance among females and those who stay much at home; its attack on all ages and both sexes; the spread of the disease in new areas; its first sweep of severity in such areas, and later its gradual decrease and the relative immunity of the inhabitants; its presence along streams and in mosquito-breeding areas.

"Whether a single species of the Culicidae harbors the pellagrous parasite, as in yellow fever, or more than one, as in malaria, and what this particular species may be, is a question for the future. It will probably prove to be a rural breeding, house-living, day-biting mosquito."

F. M. S.

SAMBON (L. W.). The Causation of Pellagra: A Contribution to the Discussion on Dr. Sandwith's Paper.—*Trans. Soc. Trop. Med. & Hyg.* 1913. May. Vol. 6. No. 6. pp. 231-241.

This paper maintains that the most essential and striking feature of an endemic pellagra zone is the swiftly-flowing stream and that often pellagra and malaria exhibit an inverse distribution. "Pellagra prevails chiefly in the highlands trenched by running streams; malaria prevails in the swampy lowlands." Attention is drawn to the axiom that no spot can be a true pellagra station unless the disease is found to occur in very young children who have never left it. Mention is made of three Italian infants who were attacked between the ages of three and five months and were seen by the author. He believes that even strong, healthy people are liable to contract pellagra soon after they begin to reside in a pellagrous locality and he holds very strongly that the topographical distribution is decidedly opposed to such explanations as the deficiency of nutrition or maize-mould or the photo-dynamic theory. He mentions, but does not explain, the interesting fact that patients removed from their homes to an asylum often get a recurrence of dermatitis during the spring months, in spite of a complete change of their diet and general habits. He also wishes to make it clear that he does not now limit his theory that pellagra is insect-borne to the family *Simuliidae*, but is "inclined to extend the number of possible carriers and include certain blood-sucking midges, such as *Leptoconops*, for instance, which are now placed in the family *Chironomidae*."

F. M. S.

PERRONCITO (Aldo). *Relazione sul tema: Etiologia della Pellagra.* [Etiology of Pellagra.]—*Sperimentale.* 1913. Sept. 5. Vol. 67. Suppl. to No. 4. (Atti dell' viii Riunione della Soc. Italiana di Patologia tenuta in Pisa nei giorni 25, 26, 27 Marzo 1913.) pp. 94-139. [With Discussion pp. 139-142.]

The author gives a long historical resumé of the three theories as to the causation of pellagra—maize, toxic maize, and parasites. He sums up by pleading that all the splendid work which has been done in Italy to fight poverty and prematurely grown or diseased maize should not be discontinued, though he confesses that the etiological problem is still unsolved.

The theory of insufficient nutrition finds no favour with him, because in times of famine pellagra may be absent, and on the other hand it is sometimes met with among the affluent.

He recapitulates LOMBROSO's experiments, and says that although he did not prove that pellagra is caused by diseased maize, he did succeed in introducing an important number of humanitarian laws, and he established the fact that diseased maize is a dangerous diet. Among those who have advanced parasitic theories he specially mentions CENI, TIZZONI, SAMBON and ALESSANDRINI. He states that CENI's experiments have not been confirmed by others and he considers it a very weak point

in his theory that the spores of various *Aspergilli* were able to migrate into the internal organs of animals without developing, though the experimenter suggested that they there set up changes of a toxic character. TIZZONI's polymorphic streptobacillus could not be found by the author in a score of pellagrins and others have also failed to find it. SAMBON's views are quoted at length and praise is given to him for disregarding the validity of Italian statistics, but he considers that it is unscientific for SAMBON to have absolutely settled upon the insect carrier, before we know anything about the parasite, or even if there be a parasite. ALESSANDRINI's filaria and drinking water theory meets with no approval. He does not reject the possibility of pellagra some day being proved to be a protozoal disease and cites the success of atoxyl treatment as an argument in favour of this view.

F. M. S

TIZZONI (G.) & DE ANGELIS (G.). Studien über die Biologie und die Morphologie des pleomorphen Streptobacillus der Pellagra. [The Biology and Morphology of the Pleomorphic Streptobacillus of Pellagra.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. May 3. Vol. 69. No. 1/2. pp. 5-8.

This is a paper in continuation of one on the same subject reviewed in this *Bulletin*, Vol. 1, p. 681. The authors now maintain, as they did before, that Type A of their streptobacillus can pass into Type B. They still believe from its morphological characters that their organism should be classed in the group of *Actinomyces*. They consider that their researches throw light upon the difficult question of how pellagra may be evolved from diseased maize.

F. M. S

TIZZONI (Guido) & DE ANGELIS (Giovanni). i. Ueber den Entwicklungszyklus des pleomorphen Streptobacillus der Pellagra. [On the Developmental Cycle of the Pleomorphic Streptobacillus of Pellagra.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. July 29. Vol. 70. No. 1/2. pp. 24-29.

ii. Sul Ciclo Evolutivo dello Streptobacillo della Pellagra.—*Pathologica.* 1913. Apr. 15. Vol. 5. No. 107. pp. 225-228.

These two papers are identical and consist of a second communication on the cycle of development of the streptobacillus believed by the authors to be the cause of pellagra. They claim that their bacillus can be isolated from the blood, cerebro-spinal fluid and internal organs of acute fulminating cases, and also from the blood and faeces of ordinary cases during a period of recrudescence, as well as from diseased maize.

The presence of the numbers found during life, their elective localisation in the central nervous system, experiments with serum on pellagrins and observations on guinea-pigs and monkeys, all contribute to their belief that the true cause of pellagra has been discovered.

Their researches in the laboratory at Bologna convince them that the streptobacillus may exist in three perfectly distinct phases of development, according to the conditions of the nutritive media: (1) the lower or adaptive, (2) the intermediate or actinomycetic, (3) the higher or hyphomycetic.

The authors claim that "no one can fail to see the importance of these results, from which it is certain that the pleomorphic bacillus, obtained from the blood and faeces of pellagrins, is in its various types nothing more than an adaptive form of a parasite, which, in the higher stages of its development, must be included among the higher hyphomycetes."

F. M. S.

ALESSANDRINI (G.) & SCALA (A.). *Contributo nuovo alla Etiologia e Patogenesi della Pellagra.*—*Policlinico*. Sez. pratica. 1913. June 8. Vol. 20. No. 23. pp. 805-812.

The two Roman professors have apparently relinquished the filarial theory and now consider that pellagra is strictly limited to those districts where the drinking water comes from a clay soil and that the chronic intoxication is caused by silica in colloidal solution in certain waters. They ingeniously suggest that rain water passing over clay or aluminium silicate causes hydrolysis, with the formation of aluminium hydrate and silicic acid. These in the colloid state, being incompatible, precipitate one another, leaving an excess of silicic acid in the water. The first effect of this solution of colloidal silica upon animals is a retention of sodium chloride.

The colloidal silica combines with the salt and transfers it to the proteid molecule where a new reaction takes place by hydrolysis, producing an unstable compound which splits off hydrochloric acid, thus occasioning a mineral acidosis which is considered responsible for many pellagrous symptoms.

Experiments were made with a solution of colloidal silica, derived from the water of a pellagra zone, introduced into food and also intraperitoneally and subcutaneously into rabbits, guinea-pigs, dogs and monkeys. [The number of animals used is not given]. The symptoms produced were diarrhoea, excitement followed by nervous depression, and, in the worst cases, spastic paralysis, retention of urine, loss of weight and death with convulsions. The most notable post-mortem appearances were erythema, distended stomach, hyperaemia of most organs and transparent distended intestines, with haemorrhagic spots in the colon. The authors argued that an acidosis ought to be cured by an alkali and they therefore injected into one of their infected dogs, a solution of tri-sodic citrate 5 per cent., after which the animal regained its health and after 16 days excreted normal urine.

They then subjected nine pellagrins to similar daily injections beginning with 5 per cent. and continuing with 10 per cent. They chose individuals who had lately shown very decided symptoms of pellagra, and allowed them to continue living in



their homes, with their accustomed food and work. The patients reported that their digestive disturbance became less, appetite and strength increased, and their weight also. [No details of weight given.]

F. M. S.

#### CLINICAL AND TREATMENT.

Box (Charles R.). *Fatal Pellagra in two English Boys.*—*Trans. Soc. Trop. Med. & Hyg.* 1913. Apr. Vol. 6. No. 5. pp. 149-156: and *Brit. Med. Jl.* 1913. July 5. pp. 2-4. With 1 coloured plate.

An English boy aged 8, who had lived always in the neighbourhood of London, was admitted to St. Thomas's Hospital in September 1912, at his parents' request, because they feared that he was developing an obscure nervous disease similar to one to which another child had already succumbed.

The patient was thin and had suffered for three months before admission from odd seizures during which his body became rigid and head was stiffly retracted, but consciousness was not lost. His gait showed a mixture of spasm and ataxy, a coarse nystagmus was always present, all the deep reflexes of his limbs were increased, tenderness was elicited by firm pressure near the upper dorsal vertebrae, and he suffered from incontinence of both urine and faeces. The tip and edges of the tongue were slightly denuded of epithelium and there was an inflammatory condition of the skin around the anus. A typical pellagrous symmetrical rash was present on the backs of the hands and wrists, the orbital regions and bridge of the nose, the nape of the neck and backs of the ears and over the olecranon processes. The rash had a bright red appearance, which in hospital gradually became brownish. Box diagnosed the case from its resemblance to pictures in books and the rash is well shown in an accompanying sketch by TERZI. The patient died four weeks after admission and developed slight mental delusions during the last days of his life. At the post-mortem examination no gross changes of importance were seen. The small intestine contained much mucus and the edges of the ileo-caecal valve were purple and superficially ulcerated. A culture made from the blood immediately after death showed the presence of *Streptococcus pyogenes*. Box is now certain that the other child who was an in-patient at St. Thomas's Hospital for a fortnight in September and October 1910 and who died at home in January 1911 was also a case of pellagra. This diagnosis was not considered at the time and it was thought that the boy was suffering from an anomalous form of combined degeneration of the cord in some respects resembling Friedreich's ataxy. The mother now says that this boy also had a rash, which was present only in the summer and occurred during the last three or four years of life. This earlier case was born in Ireland but had always lived, since infancy, in the vicinity of London.

F. M. S.

HAMMOND (J. A. B.). **A Case of Pellagra in England, probably contracted in Scotland.**—*Brit. Med. Jl.* 1913. July 5. p. 12.

The symptoms, not usually seen in pellagra, which require explanation are: coated tongue, abdominal pain, tenderness, and a slight evening temperature which all disappeared after the appendix was removed; a second phase of acute abdominal pain, accompanied this time by offensive stools, mucus and profuse haemorrhage, all of which yielded to vaccine treatment; a dermatitis which became suppurative about the lips and between the fingers; ulceration of the tongue and mouth; a return of abdominal pain and tenderness, "worse just before and after the bowels acted," necessitating laparotomy. The points in favour of the diagnosis of pellagra are: dermatitis of face, wrists and hands occurring in the second spring of the illness, heartburn, emaciation, exaggerated knee jerks and some mental confusion before death. Permission was only obtained to examine the abdomen after death and this threw no light upon the diagnosis. [The author's reasons for supposing that this was "undoubtedly" a case of pellagra are not very apparent. The patient was under observation for two years until her death in the Isle of Wight, but during that time she does not seem to have been seen by any physician acquainted with pellagra.]

F. M. S.

SAMBON (Louis W.). i. **The Natural History of Pellagra. With an Account of Two New Cases in England.**—*Brit. Med. Jl.* 1913. July 5. pp. 5-12.

ii. **Pellagra in Great Britain: Three New Indigenous Cases.**—*Ibid.* July 19. pp. 119-120. With illustrations.

iii. **Pellagra in Great Britain. Notes of some Further Cases.**—*Ibid.* Aug. 9. pp. 297-298.

i. The first case mentioned is that of Dr. Box, "the first indubitable case recognized in England. The second is a kitchen gardener of Lymington who has never been out of Hampshire; he suffered from a symmetrical summer rash, vertigo, tremor of hands, depression of spirits, bodily weakness and diarrhoea. He stated that he had sometimes in the evenings been bitten by small black flies while gardening. The author visited the Lymington river and many of its tributary streams and found in all of them larvae and pupae of *Simuliidae*. His assistant also found similar larvae and pupae in streams near the house where Box's patient lived. The author visited this house and was rewarded by finding a third boy of the same family who had a slight rash but no other symptoms of pellagra. This first paper is enriched by a description of *Simulium* and by drawings of the insect larva and pupa.

ii. The second paper describes (1) the fatal case of a child in Shropshire and the existence of small swarms of *Simulium* near a brook where the child had frequently played; (2) the reported case of a woman from Cardiganshire; (3) BLANDY'S fatal case in the Napsburg Lunatic Asylum.

"With regard to the etiology of pellagra, the British cases absolutely disprove the maize theory of its causation."

iii. The third paper describes two cases at the Prestwich County Asylum, both women, and another woman from Walthamstow which was posthumously diagnosed by Dr. Raymond CRAWFORD, who had treated her at King's College Hospital in 1912-1913.

[It is earnestly to be hoped that medical officers of lunatic asylums and also practitioners in rural districts will keep a sharp look out for possible cases.]

F. M. S.

BLANDY (Gurth S.). *A Contribution to the Study of Pellagra in England.*—*Lancet*. 1913. Sept. 6. pp. 713-717. With 2 plates.

This paper, by the Assistant Medical Officer of the Middlesex County Asylum, suggests that pellagra is not so rare a disease in English lunatic asylums as has been supposed. Three of the eleven cases briefly described were visited by Dr. SAMBON who confirmed the diagnosis of pellagra, while eight others have been brought forward from past or present records of the Asylum. This is similar to the early history of the disease in the United States, for after the diagnosis of the early insane patients, many former cases of obscurity were resuscitated and thought to be undoubted instances of pellagra undiagnosed during life.

Case 1, who had never lived far from London, developed in June 1913, a month after her admission, dermatitis of face, wrists and dorsal aspects of hands and fingers; it was noted that the terminal phalanges were less affected than the rest of the hands. She died on July 29th after being in an afebrile "typhoid state" for three weeks. The results of the pathological investigation by an expert will be published later. This patient "was not out in the open at any time while in this hospital, but was on one occasion only under a verandah which was protected from the sun."

The plates accompanying the article show twelve photographs from four cases, the skin lesions depicted looking exactly like the well-known pellagra eruption.

Case 7 was admitted when she was 37 years old and had already been insane for 20 years. In the summer of 1909, about eighteen months after admission, she developed dermatitis of the hands (called sunburn), stomatitis, diarrhoea and increased mental excitement, followed later by causeless vomiting and emaciation. Eventually there were noted albuminuria, subnormal temperature and increasing feebleness till death on November 17th, 1912.

"At the post-mortem examination very marked chronic nephritis, some pulmonary infarcts and a small, evidently terminal, empyema were found."

Of the eleven cases ten were females, the remaining one being a doubtful case of a male who died suddenly and was found post mortem to have had pulmonary tuberculosis. All the patients, except one, were adults, aged from 20-40 years.

"A very striking feature of the cases described in this article is their severity." This is another similarity with the early

American cases, for in Egypt and Italy it is rare to meet with fulminating cases.

[This paper is well worth perusal. Case 11 became rapidly worse and died on September 15th, 1913.]

F. M. S.

COLE (J. W. E.). *Notes of a Case of Pellagra.*—*Lancet.* 1913. Sept. 6. pp. 717-718.

This is a case reported from the Bethnall House lunatic asylum in London, which was visited by SAMBON and STURLI of Trieste who corroborated the diagnosis. A somewhat symmetrical rash on the face, neck, hands and wrists, followed by desquamation, diarrhoea, exaggerated knee jerks and skin reflexes, were the symptoms present. Mentally a condition resembling katatoniac stupor existed.

F. M. S.

DEEKS (W. E.). *Pellagra in the Canal Zone: Its Etiology and Treatment.*—*Southern Med. J.* 1913. July 1. Vol. 6. No. 7. pp. 438-446.

This is a report of 30 cases treated in Ancon Hospital, including the progress of 12 cases previously recorded.

Gastro-intestinal symptoms were present in 77 and dermatitis in 83 per cent. Evidence of nephritis existed in 83 per cent.; "some showed chronic changes, others those of acute conditions which rapidly subsided on treatment." The author repeats his former belief that pellagra "is the result of an auto-intoxication caused by the action of some ferment or organism on a carbohydrate diet to excess, to the exclusion of green vegetables and fruits during the warm weather when metabolic activity is lessened."

He asserts that "there is no endemic center." [This unusual assertion arises apparently from the fact that most of his patients came from different islands, Martinique, Guadeloupe, Trinidad, Jamaica, Barbados, Montserrat and St. Lucia.] He considers that maize, when consumed largely, is probably an important factor, but when taken sparingly it is of no more importance than any other starchy food. He believes that the increase of pellagra is partly due to the inordinate consumption of cane sugar in all civilized countries, for instance in the United States, of 3.28 ounces daily per head, and that pellagra is the result of auto-intoxication. His treatment is to avoid sugar and starchy food in every form, to limit the diet of pellagrins to fresh fruit juice, preferably orange, with broths and milk, and after a few days, to a carbohydrate-free regimen. He has also great faith in dilute nitric acid, 15 to 30 drops in three quarters of a tumbler of water three times a day before meals.

F. M. S.

VON PROBIZER (Guido). **Praktische Bemerkungen zur Diagnose der pellagrösen Hautveränderungen.** [Practical Notes on the Diagnosis of Skin Lesions in Pellagra.]—*Dermatol. Wochenschrift.* 1913. June 7. Vol. 56. No. 23. pp. 637-649. With 1 plate.

The author writes from a pellagra asylum in the Austrian Tyrol and agrees with LOMBROSO that the skin manifestations of pellagra are not the most important symptoms of the disease. In other words, the presence of erythema is of great use in diagnosing a doubtful case, but its absence does not justify one in regarding a case as not pellagrous. His asylum has since the beginning of this century been able to furnish clinical material to the psychiatric and dermatological clinics of Innsbrück, in the latter of which Professor MERK prepared his well-known casts.

Unable to accept the optimistic statement of LUSSANA, that the fight against pellagra will soon fail in its objective, because the material for further study will be lacking, the author recognizes that the mass of material with which he had to deal ten years ago at Trentino can no longer be found, and that pellagra is now assuming a modified form with partial disappearance of the skin lesions. He pleads for a more thorough histological and pathological study of the skin lesions and points out that the country doctor has now great difficulty in diagnosing between pellagrous erythema and other forms of erythema on the back of the hands said to be due to the sun or to alcohol, which occur in a district where pellagra is endemic. He quotes at length the discussion at the Bergamo pellagra congress and the opinions of the Venetian dermatologist, Professor FIOCCO. The plate shows the dorsal regions of the hands of a patient.

F. M. S.

BARDIN (James C.). **Further Observations on the Blood of Pellagra.**—*Amer. Jl. of Insanity.* 1913. July. Vol. 70. No. 1. pp. 155-159.

The author having already published observations made on fourteen cases of pellagra, this is the result of the study of seven additional cases in negroes of Virginia. The author finds that he can by blood examination discriminate between (1) uncomplicated pellagra, (2) pellagra associated with tuberculosis, and (3) pellagra complicated by intestinal parasites, such as *Ascaris*, *Trichocephalus* and *Strongyloides intestinalis*.

Two slides were examined at each of four counts and at least 600 corpuscles were averaged. In (1) polymorphonuclears were reduced, small and large lymphocytes increased, eosinophiles normal. In (2) polymorphonuclears somewhat increased, small lymphocytes somewhat reduced, large lymphocytes slightly increased, eosinophiles normal. In (3) polymorphonuclears much reduced, small and large lymphocytes increased, eosinophiles greatly increased. It is believed that the small lymphocytes increase in proportion to the severity of the skin lesions.

[No percentages are given.]

F. M. S.

**NILES (George M.).** *The Role of Hydrotherapy in the Treatment of Pellagra.*—*Amer. Jl. of the Med. Sciences.* 1913. Aug. Vol. 146. No. 2. (No. 497.) pp. 230-233.

The author, who is Professor of Gastro-enterology and Therapeutics in Atlanta (Georgia), after an experience of 75 cases, claims that energetic hydrotherapy should be employed in addition to other methods of treatment. He recommends for nausea the drinking of two to six glasses of tepid water daily, unless lavage can be performed by an expert. The frequent diarrhoea may be greatly alleviated by hot colon irrigations, followed by cold sitz baths for five to ten minutes; this double method may be repeated two to four times daily when the patient is not too weak. Cold abdominal compresses, various hot packs, saline baths, "revulsive compresses to the spine," cold percussion spinal douche baths, beginning with a temperature varying from 45° to 70°, all are praised. "Cold packs are seldom indicated, though in some 'typhoid' cases, with muttering delirium, they hold a doubtful place."

[General and local saline baths have been used for many years in some hospitals in Italy. They give temporary relief.]

F. M. S.

**KELLY (D. W.).** *A Case of Pellagra treated with Salvarsan.*—*New Orleans Med. & Surg. Jl.* 1913. Aug. Vol. 66. No. 2. pp. 106-107.

A young white woman of Louisiana began to suffer from vomiting in the autumn of 1910 and a year later developed mental symptoms. In April, 1912, she showed for the first time the "skin lesions of pellagra" and two months afterwards she applied to the author for treatment. She was then "ravingly insane and nothing but skin and bones, weighing about 75 pounds." He gave her intravenously three grains of salvarsan and increased the dose every 10 or 15 days until he had given a fifth dose of nine grains seven weeks from the beginning of the treatment. She began to gain weight and show a general improvement from the very first dose, and the author states that in the autumn her mind was "perfectly restored" and she weighed about 175 pounds. For diet she was not allowed maize products and was given as much milk and eggs as possible.

F. M. S.

**SHOEMAKER (Harlan).** *Pellagra, Surgery, The Colloids and Strong Drugs. Also Introducing a Possible New Etiological Factor.*—*New York Med. Jl.* 1913. Aug. 2. Vol. 98. No. 5. pp. 214-219.

The writer says that in the year 1910 there were known to him only about ten cases of pellagra in Cleveland County, North Carolina; in 1912, there were 100 cases and in 1913 the incidence "is almost that of measles." Early surgery is now recommended. "I have deliberately opened the upper abdomen in two early cases of pellagra for the purpose of draining the gall bladder,

and I was fortunately able to remove the appendix through this high incision." Both patients were discharged from the hospital on the 21st day. One had an uneventful recovery, the other, a woman, became pregnant and nearly died "from recurring intestinal symptoms" followed by abortion.

The author's experience is that for the first six to nine months pellagra "remains as a mild catarrhal indigestion confined to the upper abdomen." Because the gall bladder is sometimes a reservoir for microbes, he considers it possible that the pellagra germ may lurk there. "Gall bladder drainage is the operation of election, because it is as high in the alimentary tract as it is necessary to drain, and, being an operation of election, should be accompanied by an appendectomy." He goes so far as to state that "one in twelve pellagrins are surgical and can be treated by drainage." According to him, all authors agree that pellagra is a disease of hypoleucocytosis, and therefore he argues that artificial leucocytosis should be produced by a surgical operation, or by administering colloids intravenously. He is, however, aware of the danger of alcohol, morphia and pregnancy.

F. M. S.

**LAW (William Lamar).** Treatment of Pellagra with Lactic Acid Bacilli.—*Jl. Amer. Med. Assoc.* 1913. July 5. Vol. 61. No. 1. p. 27.

This is a commendably short paper of six lines, in which the writer states that he has treated his last five cases of pellagra with tablets of lactic acid bacilli, followed by an improvement in their subjective symptoms.

F. M. S.

#### EXPERIMENTAL AND PATHOLOGY.

**HARRIS (William H.).** The Experimental Production of Pellagra in the Monkey by a Berkefeld Filtrate derived from Human Lesions. A Preliminary Note.—*Jl. Amer. Med. Assoc.* 1913. June 21. Vol. 60. No. 25. pp. 1948-1950. With 2 figs.

In the spring of 1910 an autopsy was made on an uncomplicated case of pellagra, two hours after death. Portions of the spinal cord, skin and digestive tract were mixed with equal amounts of normal saline solution, ground together in a mortar and after coarse filtration the juice was passed through a Berkefeld filter, letter N. The filtrate was then injected subcutaneously and intracranially, in large quantities into a healthy monkey (*Macacus rhesus*), which remained "normal for many months" and then developed irregular dark patches on the face, forearms, hands, back and sides of the body. The monkey gradually grew thin and weak and died. Post mortem were found some inflammation of the small intestine and, microscopically, "extensive hyperkeratoses of the epidermis, marked increase in the depth of the papillae and abundant pigmentation in the deeper portions of the corium where many chromatophores have wandered into the deeper strata." [The monkey's spinal cord was apparently not examined.] Sceptics did not accept this

as a case of monkey pellagra, so it was determined to wait until a similar typical and uncomplicated fatal human case should be available. On December 2, 1912, a similar monkey was inoculated as above, and this second monkey remained apparently normal for two months. On February 12, 1913, Monkey 2 was given a second injection from the brain, spinal cord, intestinal and skin lesions, obtained direct from a human autopsy of pellagra. Early in May, Monkey 2 showed irregular patches of a copper or dusky red colour about the face, and the patches gradually became somewhat butterfly-shaped. Later symmetrical copper coloured lesions appeared on the summit of the concha of both ears and at the external canthus of the eyes and also over the backs of the hands. The monkey became melancholic, emaciated and weak, with diminished appetite, diarrhoea, salivation and granular tip of the tongue. On June 1, when the author wrote his paper from the Laboratories of Tulane University, Monkey 2 was evidently dying. Also on February 12, 1913, Monkey 3 was injected intravenously and intracranially with filtrate prepared from the brain and cord of the human case. (On June 1, "Monkey 3 is still in good condition and quite lively but presents about the face a few irregular, rust-coloured macules, quite like the early lesions of Monkey 2." [The two illustrations are of Monkey 2, and are suggestive of pellagra. We cannot draw any conclusions, until a larger number of animals have been inoculated. The introduction of pellagra in this way into monkeys, if accepted, will be the strongest argument yet advanced that pellagra is a protozoal disease.]

F. M. S.

DEARMAN (W. A.). **Pellagra induced in a Monkey. Preliminary Report.**—*Mississippi Med. Monthly*. 1913. Mar. Vol. 17. No. 11. pp. 220-221.

The author states that he has induced "a typical case of pellagra in a monkey" and he seems to have exhibited the sick monkey to the Harrison County Medical Society. The animal is said to have suffered from diarrhoea, emaciation and desquamation of dorsum of hands, lower part of forearms, elbows and root of the tail. [Dr. HARRIS's experiments are being imitated by many in the United States. This paper can hardly be taken seriously for the author does not tell us what he did nor when he he did it.]

F. M. S.

NICHOLLS (Lucius). **The Pathological Changes in Pellagra and the Production of the Disease in Lower Animals.**—*Jl. of Hygiene*. 1913. July. Vol. 13. No. 2. pp. 149-161. With 2 plates.

The microscopical sections, illustrated by the plates, were made in the Quick Laboratory, Cambridge, but the rest of the work was done in St. Lucia. An analysis is given of eight autopsies of mulatto pellagrins, all of whom had suffered during life from



typical eruptions, emaciation, irritable dementia, soreness of mouth and persistent diarrhoea. There were found, some atrophy of heart; fatty degeneration of liver with shrinkage of weight to an average of 37 and a half ounces; shrinkage of spleen to an average weight of three ounces (one case seven drachms), which is the more important because the patients had all come from malarial localities; thinning of walls of stomach and intestines; atrophy of the stratum corneum of skin. "In some cases the dura mater was thickened, and the pia mater showed irregular areas due to thickening. The convolutions of the brain were atrophied, and were of firmer consistence than normal . . . . There was an increase in the fibrous tissue in the meninges and the arterioles supplying them were thickened, also the endothelium of the capillary vessels showed degeneration with a tendency to proliferation. The cortical cells of the brain were shrunken and degenerate." The author considers that the pathological changes are greatly due to degeneration of the capillaries, giving rise to stenosis and blood stasis, and consequent leakage from the weakened vessels. He suggests that the eruption on exposed skin surfaces is caused by the solar radiation and possibly other factors accentuating the damage which has taken place in the vascular supply. With regard to the sand-fly protozoal hypothesis he states that the evidence of SAMBON and CHALMERS "in support of the theory is of a very frail nature."

He is of opinion that previous experimenters on animals and poultry have expected too much in hoping to reproduce "the exact picture or all the signs and symptoms which occur in man. If, however, it can be shown that a certain organism or a material produces an analogous condition leading to the death of the animal, and the post-mortem and microscopical examination show similar changes, evidence of the cause of the disease in man is afforded."

He experimented upon six rats by giving them in their food "various preparations of decomposing corn-meal"; three died between the 8th and 31st day, two were killed on the 34th day, and the sixth escaped. Four control animals remained healthy. Four of the five dead rats showed fatty degeneration of liver and numerous haemorrhages among the liver cells, also an increase in the fibrous tissue of the spleen, numerous haemorrhages and a degree of pigmentation of spleen "which exactly compared with that which is seen in sections from human subjects who have died of pellagra."

Though the advanced arteriole thickening and extreme atrophy and cellular degeneration of chronic human cases were absent in the rats, the author is of opinion that "these preliminary experiments show that corn-meal acted upon by various micro-organisms develops toxins which may produce in animals a condition analogous to pellagra in man." [These experiments are too few in number to prove much. It is not clear what food the control rats were given.]

Morr (F. W.). The Histological Changes in the Nervous System of Dr. Box's Case of Pellagra, compared with Changes found in a Case of Pellagra dying in the Abassieh Asylum, Cairo.—*Trans. Soc. Trop. Med. & Hyg.* 1913. Apr. Vol. 6. No. 3. pp. 157-160. With 12 figs: and *Brit. Med. Jl.* 1913. July 5. pp. 4-5. With 3 plates.

This paper, like that of Dr. Box, was read before the Society of Tropical Medicine and Hygiene in February, 1913. It deals with the case of the patient diagnosed during life by Dr. Box and is accompanied by photomicrographs of the sciatic nerve, spinal cord and cerebellum. The material sent to the author for examination consisted of the brain, spinal cord, a few of the posterior spinal ganglia and a piece of the sciatic nerve. The Marchi method showed the following changes in the fibres: recent scattered degenerative fibres in all the longitudinal and transverse sections of the sciatic nerve, similar changes in the roots of the *cauda equina* and throughout the white matter of the spinal cord, more marked in the postero-lateral and posterior median columns than elsewhere. The Weigert-Pal method displayed general diffuse sclerosis throughout the white matter of the cord everywhere, while the naked eye could see sclerosis affecting the direct cerebellar and Gowers's tracts, Goll's column and the crossed pyramidal tracts. Descending degeneration could be seen in the lower lumbar and sacral regions. Some scattered sclerosis was also observed in the pyramids of the medulla. In none of the sections made was there evidence of meningeal or peri-vascular infiltration with lymphocytes or plasma cells or with polynuclear leucocytes. "The absence of chronic meningo-encephalitis and meningo-myelitis, so characteristic of protozoal diseases, contraindicates the protozoal theory of the origin of pellagra, yet it does not disprove it, for in malaria the vessels may be filled with the organisms and yet no inflammatory peri-vascular and meningeal reaction is seen; moreover, although all the changes are like those produced by a chronic toxæmia, yet the cause of that toxæmia has not been satisfactorily determined."

Varying degrees of chromatolysis were seen, with swelling of the cells, disappearance of the Nissl granules, except at the periphery, and frequently eccentric position of the nucleus. Comparison of the changes in the cells with those of the Cairo lunatic, who died from pellagra in the asylum, showed no essential difference. "There is a combined sclerosis of the spinal cord more marked in the Egyptian case; there are the same changes in all the ganglion cells of the spinal cord and brain and there are no signs of acute or chronic vascular or meningeal inflammatory changes." [This report is a model which should be read and imitated by all those who have the opportunity of conducting autopsies upon the pellagrous in well-equipped hospitals.]

CENTANNI (E.) & GALASSI (C.). **Sul Doppio Effetto, Tossico e Unilaterale, dell' Alimentazione Maidica.** [On the Double Action, Toxic and One-sided, of Maize Food.] — *Sperimentale*. 1913. Sept. 5. Vol. 67. Suppl. to No. 4. (Atti dell' viii Riunione d. Soc. Italiana di Patologia tenuta in Pisa nei giorni 25, 26, 27 Marzo, 1913.) pp. 142-150.

Recent chemical researches with reference to beriberi and scurvy stimulated the authors in the Institute at Siena to experiment on the nutritive value of maize. They found that three guinea-pigs fed on maize in artificial darkness died in 14 to 29 days, after loss of weight, failure of appetite, diarrhoea, hair falling off, with a little redness, oedema and desquamation of the skin of the feet. After death there was seen congestion of the gastro-intestinal tract and in one of the stomachs there was a clot of blood.

Three other guinea-pigs kept similarly but fed also on vegetables during 51 to 75 days gained in weight and health and showed no skin affection. In the second experiment three guinea-pigs were fed upon maize extract, treated by ether or alcohol, and were exposed to light and for part of the day to the sun. All died in from six to ten days, after refusing to eat. Two other guinea-pigs treated in the same way but given vegetables in addition lived for 40 days in the best possible health; they were then gradually deprived of their vegetable ration and succumbed 23 and 25 days after it was stopped. At the autopsy of one of them were seen typical signs of experimental scurvy, as described by HOLST and FRÖLICH, looseness of teeth, haemorrhages in intestines and pancreas and changes between the shafts and epiphyses of femurs and ribs.

Other experiments were tried on rats which stood the exclusive diet less well than guinea-pigs. The authors consider that in maize nutrition the factor of exclusive (one sided) diet is of greater importance than the photo-dynamic factor.

F. M. S.

CASSAMALLI (Ferdinando). **Sulla Persistenza del Potere Vitale di Spore Eumicetiche, esposte ad Alta Temperatura.** [Persistence of Vitality of Certain Spores of Mould, after Exposure to a High Temperature.]—*Riv. Pellagologica Italiana*. 1913. July. Vol. 13. No. 4. pp. 51-54.

A note from the laboratories of Reggio Emilia. The author finds that the heat to which maize flour is subjected in making *polenta* does not kill the spores of certain moulds which are apt to pollute the flour eaten by pellagrous people. The *Aspergillus fumigatus* has a greater resistive power to heat than some varieties of *Penicillium*.

[There is nothing new in this, for it was maintained years ago by CARR.]

F. M. S.

FINATA (L.) & NOVELLO (F.). *Ricerche sulla Ipersensibilità dei Pellagrosi.*—*Pathologica*. 1913. Aug. 15. Vol. 5. No. 115. pp. 492-493.

A note from Verona on the reaction seen in pellagrins after the intramuscular injection of sterile extracts of damaged maize.

Some pellagrogenin\* having been procured from Professor VOLPINO, 41 pellagrins each received one intramuscular injection in the buttock. A positive reaction took place in 31, negative in nine and doubtful in one case. An intense reaction was only seen in those who had recently been admitted to the sanatorium and was chiefly confined to the nervous system. The authors consider that four of their cases also showed an increase of erythema. They maintain that eighteen healthy people similarly injected showed no reaction and [on these very slender results] they state that pellagrins have really this hypersensitivity.

F. M. S.

THOMAS (W. Rees). *Pellagra and Drug Intoxication*. [Correspondence.]—*Lancet*. 1913. Sept. 13. p. 842.

This writer, from the East Sussex Asylum, is surprised to hear that most cases of pellagra in England are lunatics in asylums. But in the United States the early patients were chiefly discovered in such institutions, and even in Italy to-day a visitor is more likely to be shown pellagrous cases in an asylum than in a general hospital. He suggests that sulphonal or trional may produce a symmetrical rash, resembling pellagra, accompanied by diarrhoea and emaciation.

[It is to be hoped that he will now publish photographs or sketches of these drug rashes, which, from his description, are evidently somewhat different to the scarlatiniform eruption or generalised erythema which are well known to betray sulphonal poisoning. He does not state whether haematoporphyrinuria was present in his cases.]

F. M. S.

\* See *Pathologica*. 1913. Mar. 15. Vol. 5. No. 105. pp. 174-176.

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## LEPROSY.

## HISTORY.

MORROW (Robert). *History of Leprosy in South Africa up to the Establishment of the First Leper Asylum, Hemel en Aarde.*—*S. African Med. Rec.* 1913. May 24. Vol. 11. No. 10. pp. 174-183.

The Bushmen, a race of pigmies, inhabited Africa south of the Zambesi for 25 centuries, for so the erosion of the igneous rocks on which their drawings were executed would testify. They were a nomad people existing on ant-eggs, roots, and the produce of the chase. It is probable that leprosy was endemic among them. The extensive ruins of Zimbabwe and the old gold-workings scattered throughout southern Rhodesia indicate that there was a large foreign population at one time, by which also the infection might have been introduced. In the 15th century the Bushmen were invaded by the Hottentots, and afterwards by the warlike Bantus. Soon after the advent of the European in the 17th century their extermination was completed. Both Hottentots and Bantus were of leprous stock in all probability.

The first authentic record of leprosy occurs in the Archives of the Cape of Good Hope in the year 1756. The presence of leprosy in European families was then reported officially. At the beginning of the 19th century the disease was rife, especially among the Hottentots. In 1817 a leper asylum, Hemel en Aarde [Heaven on Earth], was established, and accommodated 100 natives who were placed under no restrictions. It was situated in Caledon, and thither all the lepers of the colony were transferred.

C. Birt.

KEUSSEN (Herm.). *Beiträge zur Geschichte der Kölner Lepra-Untersuchungen.* [The History of Leprosy Inspections in Cologne.]—*Lepra.* 1913. July. Vol. 14. No. 2. pp. 80-112.

Short clinical notes are given of 178 persons who were examined for leprosy by the Faculty of Medicine of Cologne in the years 1491-1664. Biographical accounts of the Members of the Faculty are appended, and also official documents concerning leprosy in Cologne and its vicinity, 1357-1712.

C. B.

ASEN (Johannes). *Eine Leprosenordnung von Melaten bei Köln aus dem 16. Jahrhundert.* [Leprosy Regulations at Melaten near Cologne in the 16th century.] — *Lepra.* 1913. July. Vol. 14. No. 2. pp. 70-72.

The leper house at Melaten near Cologne dated back from the 12th century, and was the oldest and most important of those situated on the Lower Rhine. The instructions which were given to the officials and the certificate which had to be signed by the examining authorities are inserted *in extenso*.

C. B.

CLIPPINGDALE (S. D.). *The Leper Window.*—*Leprosy*. 1913. July. Vol. 14. No. 2. pp. 78-79.

Leper windows are found in 13th and 14th century churches of England and Normandy. Their usual position is the south wall of the chancel. In the Roman Catholic Office for the seclusion of a leper, he is forbidden to enter churches, and in an old fresco in Eton College Chapel a priest is handing something to a person kneeling outside. Recently it has been suggested that these windows were not designed for the use of lepers, but the author on reviewing the evidence concludes that the windows were made for the benefit of some persons who were entitled to the offices of religion, but whose presence in the church was not permitted.

C. B.

#### DISTRIBUTION.

*La Lèpre dans les Colonies françaises.*—*Gaz. des Hopit. Civils & Militaires*. 1913. July 31. Vol. 86. No. 86. pp. 1396-1397.

Leprosy is unevenly distributed in Indo-China. It is estimated that there are 4,000 to 5,000 lepers in Tonkin, 5,000 to 6,000 in Cochin China, and a few only in Annam.

In the year 1903 it was enacted that a leper asylum should be established at Culao-Rang in Cochin China, and that lepers should be segregated. In 1909 lepers were prohibited from parading the public streets, from occupying public offices, and from following certain callings.

Leper villages, situated 2 kilometers from the healthy population, are preferable to large leper asylums, such as that at Tre-Truong, 11 kilometers from Hanoi, where 986 lepers are accommodated. At the end of 1912 there were twenty of these leper colonies in existence. Leprosy is no longer on the increase in Indo-China.

C. B.

BLUE (Rupert). *The Public Health Aspects of Leprosy in the United States.*—*Jl. Amer. Med. Assoc.* 1913. Sept. 20. Vol. 61. No. 12. pp. 943-946.

It is probable that leprosy was introduced into the United States from abroad. In the year 1901 there were 278 lepers, of whom 145 were born in the United States; only 72 were segregated. In 1912 the number was reported to be 146 in a population of 90 millions; all these lepers with a few exceptions were being treated in special institutions.

The disease is chiefly confined to the Atlantic seaboard, the Gulf Coast, the Pacific Coast, and the north central States, where it is on the wane. There are 696 lepers in Hawaii, where no cases were known before 1838; 2,754 in the Philippine Islands; and 28 in Porto Rico.

Leprosy is notifiable in 18 States. The Immigration laws forbid the landing of lepers, and provide that an alien leper may be deported within 3 years of his arrival. Railway companies

are not allowed to transport lepers from State to State. The author advocates the establishment of a National Leper Home.

In the discussion which followed Dr. BRACKEN stated that in the year 1900 there were 27 lepers in Minnesota, now there are 13 only. Seven were born in Minnesota. They are not segregated.

C. B.

#### TRANSMISSION.

LEBOEUF (A.). *Notes sur l'Epidémiologie de la Lèpre dans l'Archipel Calédonien.*—*Bull. Soc. Path. Exot.* 1913. Oct. Vol. 6. No. 8. pp. 551-556.

The author has been occupied for two and a half years in ascertaining if insects and acari play a part in the transmission of leprosy. Four or five per cent. of the 3,400 inhabitants of the coral island, Maré, in the Loyalty group, are lepers. There is no water on the island; hence there are no Simuliidae, Chironomidae, Anophelines, and very few other mosquitoes. In that part of the island of Lifou where mosquitoes are numerous, there is no leprosy, though it prevails elsewhere. It is improbable, therefore, that any of these flies carry the infection. Pediculi are not incriminated, for the men who are less verminous than the women are infected more frequently. Laboratory experiments with fleas and bugs are negative. Leprosy bacilli may sometimes be found in comedones which contain the demodex, and the author has already reported the presence of *B. leprae* in house-flies (see this *Bulletin*, Vol. 1, p. 558). He thinks that both may transmit the disease, but that the most common mode of infection is by the conveyance of the bacillus from person to person, or by contaminated articles. The zone of dissemination of the bacilli is narrow, for family infection is very frequently observed in New Caledonia. In his opinion the daily use of soap and water is one of the most important preventive measures. Tubercular leprosy, which at one time was the form observed in the large majority of cases, is being replaced by mixed, anaesthetic, and abortive leprosy.

C. B.

THOMSON (David). *Preliminary Note on Bed-Bugs and Leprosy.*—*Brit. Med. Jl.* 1913. Oct. 4. p. 849.

Neither 105 bugs fed on lepers in Liverpool, Panama, and Trinidad, and 35 caught on the beds of lepers, nor 107 bugs which served as controls, contained acid-fast bacilli when they were stained by the Ziehl-Neelsen method.

[SKELTON & PARHAM's observations are thus confirmed; see this *Bulletin*, Vol. 1, p. 559.]

C. B.

#### BACTERIOLOGY.

i. FRASER (Henry). *The Cultivation of the Bacillus of Leprosy* *Jl. Trop. Med. & Hyg.* 1913. June 2. Vol. 16. No. 11. p. 164.

ii. FRASER (Henry) & FLETCHER (William). *The Bacillus Leprae: Has it been cultivated?*—*Lancet.* 1913. Sept. 27. pp. 918-921.

i. In this *Bulletin*, Vol. 2, p. 285, is an account of Fraser's failure to grow the *B. leprae*. He now gives particulars of his

experiments with placental agar. Thirty tubes were prepared according to WELLMAN's method,\* and were inoculated with nodules of non-ulcerating leprous tissue removed under aseptic conditions from 22 patients who were free from ulcers in any part of their body. Contaminations were found in two tubes after 48 hours in the 37° C. chamber; the remaining 28 tubes remained sterile for 70 days. On this medium the tubercle bacillus grows more slowly than on glycerine-blood agar.

Thirty-five tubes of placental agar prepared according to the instructions received in a private communication from BAYON were inoculated with similar material. There was no evidence of growth in any of them after incubation at 37° C. for 85 days.

From ulcerated cases of leprosy and from post-mortem material many varieties of micro-organisms grow.

ii. The authors were working for 18 months with material derived from an institution in which were more than 250 lepers. Leper blood and normal blood media were employed both aerobically and anaerobically; no growth occurred in any of the 32 tubes though they were incubated for six months, except an early contamination in one. Thirty two serum and serum-glucose agar tubes remained sterile after inoculation with leprous nodules for 3 months. Williams' modification of Rost's medium, and Duval's amino-acid and egg albumen media were tried without success. BAYON prepares his agar from the juice of a placenta which has been frozen and thawed, by filtering it through Berkefeld and Doulton candles, and mixing the filtrate with glycerine agar. Thirty-five tubes were inoculated with leper nodules: there was no growth in 6 months.

Contaminations are visible in a few days. In some instances a crinkled brownish yellow micro-organism spread over the surface of the agar; this consisted of non-acid-fast rods among which were scattered large acid-fast coccoid spores. It is possible that some observers have mistaken such bodies for the *B. leprae*. Sometimes a saprophytic streptothrix was encountered.

#### Summary.

"Material for purposes of cultivation on various media has now been obtained from 32 non-ulcerating nodular cases of leprosy and 373 inoculations made on various culture media. It is curious, in view of the findings of other investigators, that we have consistently failed to obtain a culture of *B. leprae*. . . . From the examinations made of nodules which have been incubated on culture media for periods ranging from a few days to nine months, no evidence has been obtained that the bacilli had increased or lessened in number."

[This careful and exhaustive investigation renders it improbable that any one of the 20 or more "*B. leprae*," isolated by various observers, is the cause of leprosy.]

C. B.

\* *Centralbl. f. Bakt.* 1. Abt. Orig. 1912. Vol. 66. pp. 142-143.



**DUVAL** (Charles W.) & **HARRIS** (William H.). **Further Studies upon the Leprosy Bacillus. Its Cultivation and Differentiation from other Acid-fast Species.**—*Jl. of Med. Research.* 1913. May. Vol. 28. (New ser., Vol. 23.) No. 1. (Whole No. 137). pp. 165-198.

A medium is prepared by digesting protein for several days with trypsin, heating to 70° C. and passing through a Berkefeld candle N. and mixing with sterilized agar. [The sterility of such a medium is doubtful.] Nodules from 8 lepers were planted on this. After about a fortnight's incubation at 37° C. small colonies appeared in four of the cases. They attained their maximum in 8-10 weeks. The growth consisted of acid-fast rods. Animal experiment is not of use in distinguishing acid-fast bacteria, for the same lesions are induced by all whether they be the Timothy grass, milk, butter, smegma or the reputed leprosy bacilli. Intraperitoneal inoculation of material from the 8 lepers failed to excite the disease in the lower animals; injections of the bacillus isolated were also harmless.

There are at least 20 so-called leprosy bacilli. Levy's and Kedrowsky's cultures correspond in some respects with avian tuberculosis, in others with Moeller's smegma bacillus. Rost's and Williams' cultures are identical with Grasburger's acid-fast saprophyte. Karlinski's strain is similar to Rabinowitch's butter bacillus.

In the discussion\* on the leprosy bacillus in the section of Bacteriology at the International Congress of Medicine Duval is reported to have said that he had made several mistakes, and had stated openly that he had cultivated the leprosy bacillus, and that he now frankly admitted that he was mistaken. He denied that any one had been successful.

C. B.

**REENSTIERNA** (John). **Ueber die Kultivierbarkeit und Morphologie des Lepra-Erregers und die Uebertragung der Lepra auf Affen.** [The Culture and Morphology of the Leprosy Microbe, and the Transmission of Leprosy to Apes.]—*Arch. f. Dermat. u. Syphilis.* Orig. 1913. July. Vol. 116. No. 3. pp. 480-554. With 15 plates.

The author defines "acid-fast" as capable of retaining the stain after immersion for 15-30 seconds in a 10 per cent. dilution of nitric acid in alcohol. [The term acid-fast is usually applied to those micro-organisms which are not decolorized by long treatment with 33 per cent. nitric, or 25 per cent. sulphuric acid. SMITH and BISSET have shown that tubercle bacilli still appear red after 24 hours' immersion in 25 per cent. sulphuric acid, and leprosy bacilli up to 16 hours. Hence it is doubtful whether the bacteria which the author describes as acid-fast should be included in this class.] The material investigated was obtained from a patient who had been suffering from nodular leprosy for

5 years. [It is not stated whether it was an ulcerating case.] Streptococci were grown from the blood; these were mixed with "acid-fast" rods in subcultures which survived for four generations only. Growth appeared in all the tubes in 24 hours. Cultures of the leproma contained a streptothrix, diphtheroids, and after five days, "acid-fast" rods. By treating the mixed growth with 10 per cent. antiformin the "acid-fast" microbe was isolated in a pure condition, but it died after the second subculture.

An emulsion of the leproma was injected into the nose of a macacus; 39 days later gangrene of the part set in from which the animal died in three days. Clumps of "acid-fast" bacilli were present in the necrotic tissues. Another macacus died 59 days after an intraperitoneal inoculation of the same emulsion. Caseous deposits were found in almost all the viscera, in which were "acid-fast" bacilli. Five guinea-pigs inoculated with this material died in about a month. [The post-mortem appearances in them were those of tubercle.] For other animal experiments see the epitome of his preliminary report (this *Bulletin*, Vol. 1, pp. 194, 195). There is a good summary of previous bacteriological and experimental work, and a bibliography which occupies 6 pages. [The evidence adduced is insufficient to show that any of the bacteria isolated plays a part in the causation of leprosy.]

C. B.

ROST (E. R.). On the Leprosy Bacillus and Allied Bacilli.—*Med. Press*. 1913. Sept. 24. Vol. 147. (New ser., Vol. 96.) No. 3881. pp. 349-351.

The author insists that the cultures which he obtained from lepers before the year 1904 were those of the leprosy bacillus, because the "leprolin" prepared from them cured 7 lepers. He admits, however, that the use of this remedy was prohibited by the Government of India on the ground that contaminated cultures entered into its composition. He claims that he has isolated the *B. leprae* from the blister serum or fluid expressed from the nodules of 6 lepers. The cultures contain a non-acid-fast streptothrix which gives rise to acid-fast rods, and a non-acid-fast diphtheroid which also produces acid-fast elements. Colonies appear in 2 or 3 days when incubation is carried out at 30° C., the optimum temperature.

When these micro-organisms are injected into the peritoneal cavities of mud-fish and frogs, after an interval of 1-3 months small white vesicles stud the peritoneum, which contain acid-fast bacilli. He states that leprosy can be induced in monkeys by repeated injections of his cultures.

He now prepares leprolin from 6-week-old broth cultures of 7 micro-organisms isolated from lepers, by filtering the fluid through blotting paper and sterilizing. One to three cc. are injected into the muscles every week. A smart reaction ensues in advanced tubercular cases, but he says vaccines of the smegma

and Timothy grass bacilli cause similar symptoms. He states that chronic ulcers heal rapidly, though suppuration of the nodules sometimes occurs.

Of 30 lepers treated with this new leprolin since 1909, 4 have been cured, and improvement has been noted in many others.

[The bacteria described by Rost are not identical with *B. leprae* according to the opinion of most investigators who have studied the bacteriology of leprosy.]

C. B.

**SANTAMARIA (J. Martinez).** Acquisition of Acid-fast Properties by a Filamentary Organism cultivated from an Animal injected with a Culture of Hansen's "Bacillus."—*Jl. Trop. Med. & Hyg.* 1913. Oct. 1. Vol. 16. No. 19. p. 301.

Four weeks after the injection of Kedrowsky's micro-organism into the abdominal cavity of a mouse, nodules were found on the peritoneum. Cultures of the spleen resulted in the growth of non-acid-fast branching filaments which became acid-fast after cultivation on Dorset's medium for 14 months. Acid-fast bacilli also appeared in large numbers.

[The title is misleading since it has not been proved that Kedrowsky's culture is identical with *B. leprae*.]

C. B.

**HARRIS (Wm. H.) & LANFORD (John A.).** The Complement Fixation Test (Gay's Modification of the Besredka Method) in the Differentiation of Acid-fast Bacilli.—*Jl. of Infectious Diseases.* 1913. Sept. Vol. 13. No. 2. pp. 301-308.

The bacilli investigated were Kedrowsky's, Levy's, Clegg's, Karlinski's, Duval's, Rabinowitch's butter, Timothy grass, dung, smegma, Korn I, Grassburger's butter, and *B. phloei*. Five thousand agglutination tests were made with the sera of rabbits immunized with these acid-fast cultures. The results showed that they were of little value, and incapable of determining the identity of any with *B. leprae*, but emulsions of *B. leprae* from the tissues are not clumped by leper serum.

GAY's method of preparing antigen is to add alcohol to emulsions of the bacilli, centrifuge, and grind up the dried precipitate with such a quantity of salt that a 2 per cent. emulsion of the dried rods shall contain 0.85 per cent. of salt; 0.5 cc. of these suspensions was the dose injected into the veins of rabbits on each occasion.

#### Conclusions—

"Rabbits injected with whole bacilli or extracts of many of the members of the acid-fast group produce anti-substances of a high titre.

"The whole bacilli produce antibodies of lower potency than those produced by the Besredka antigen. This antigen produces the most potent antibodies when injected intravenously at 3-day intervals for 4 injections and the animal bled after 8 days.

"Regardless of the various methods used to produce these sensitizers, no clear-cut specificity for complement fixation has been found for the acid-fast bacilli made use of in these experiments."

C. B.

**TSURUMI (M.).** Ueber die Präzipitation und Komplementbindung mit Cuorin bei Lepra und die Beziehungen von Cuorin und Lecithin zu Lepraseren bei den Reaktionen. [Precipitation and Complement Deviation of Leper Serum with Cuorin, and the Cuorin and Lecithin Reactions compared.] — *Zeitschr. f. Immunitätsforsch. u. exper. Therapie.* 1 Teil. Orig. 1913. Aug. 5. Vol. 19. No. 1. pp. 19-30.

The sera of 36 lepers, 23 suffering from the nodular form, and 13 from the anaesthetic, were investigated. For the precipitation test a 0.1 per cent. dilution of cuorin was used. [Cuorin is a lipid allied to lecithin, belonging to the group of monaminodiphosphatides. It is obtained from the heart, traces only being present in other muscles. It is soluble in ether, but is insoluble in alcohol.] 75 per cent. of the sera were precipitated by cuorin, 20 out of the 23 nodular cases being positive, and 7 of the 13 anaesthetic lepers being negative. The complement deviation test with cuorin as antigen confirmed the precipitin test except in one instance. These reactions do not vary with the severity of the disease. The antibodies on which the reactions depend are present in the blood, pleural and pericardial fluids of the leper, but are absent from the bile. They are destroyed by heating at 63° C. for 30 minutes. The results obtained by lecithin and cuorin respectively are not always concordant.

C. B.

**SERRA (Alberto).** La Séro-Réaction de Wassermann chez les Lapins inoculés de Lèpre à la Chambre Antérieure de l'Oeil.—*Lepra.* 1912. Vol. 12. No. 3. pp. 139-146.

The complement deviation test was applied to the sera of 30 rabbits one to four months after their inoculation with human leprous tissue, or with the nodules excised in rabbits' eyes by the injection of such matter, or with Serra's bacillus (see this *Bulletin*, Vol. 2, p. 65). The result was positive in 90 per cent., when an extract of leprous tissue was used as antigen, and in 76 per cent. when a syphilitic antigen was employed. Before inoculation the sera of all but three animals were negative.

The blood of two lepers gave a positive response with an extract of the nodules of rabbits' eyes. The serum of an anaesthetic leper was negative to this antigen.

The author believes that the granuloma which appears at the site of inoculation of leprous material, or of his bacillus, in the eye of the rabbit, is a true leproma, and that his bacillus is the *B. leprae*.

C. B.

**VERROTTI (G.).** Risultati ottenuti dalle Inoculazioni intraperitoneali di Emulsione di Leproma nei Conigli. — *Giorn. Italiano d. Malattie Veneree e d. Pelle.* 1913. Mar. 28. Vol. 54. (Anno 48.) No. 1. pp. 82-91. With 1 coloured plate.

The granuloma which develops at the site of inoculation in the anterior chamber of the eye of a rabbit after the introduction of leprous material, is probably caused by the toxin of the leprosy bacilli, and not by their proliferation.

Half a cc. of an emulsion of leprous nodules, derived from a non-ulcerating case, was injected into the peritoneal cavity of each of four rabbits without result, except that the Wassermann reaction about two months later was positive in one, a leprous antigen being used.

Two cc. of the emulsion were introduced into the abdominal cavity of each of four rabbits which had been subjected to the eye experiments a year previously. In three, nodules formed in the peritoneum, liver or eye, which suggested growth of the lepra bacilli, although their presence could not be detected microscopically. The serum reaction to leper tissue extract became positive in all three rabbits.

C. B.

SPINDLER (A.). *Bemerkungen über den Komplementgehalt und die Wassermannsche Reaktion des Blutes Lepröser.* [Wassermann Reaction in Lepers, and the Amount of Complement in their Blood.] — *Dermatol. Centralbl.* 1912. Dec. Vol. 16. No. 3. p. 69.

The complement-titre of 16 lepers was not infrequently normal, but it was often reduced to one half or less, though absence of complement was not observed in any case.

MEIER noted that when 0.2 cc. of leper serum was employed in the Wassermann test a negative response was given, but when the leper serum was reduced to 0.005 cc. the reaction was positive. Spindler has confirmed this observation in seven cases. He thinks that this phenomenon is of aid in distinguishing leprous from syphilitic serum.

C. B.

## CLINICAL.

i. MARCHOUX (E.). *Etiologie et Prophylaxie de la Lèpre.*—*Bull. Soc. Française de Dermatol. et de Syphiligraph.* 1913. May. Vol. 24. No. 5. pp. 247-253.

ii. *La Lèpre.* — *Rev. d'Hyg. et de Police Sanitaire.* 1913. Aug. 20. Vol. 35. No. 8. pp. 883-939.

(i.) The matter in this paper is amplified in (ii.). In the discussion which followed, GAUCHER stated that he sees some ten lepers every year in Paris who freely associate with their fellow men. He advocates notification and isolation of lepers. Foreign lepers should be excluded.

GOUGEROT remarked that the manner of grouping of the *B. leprae* in the large lepra cells is not absolutely specific: since identical appearances may be seen in some tubercular lesions, such as tubercular ulceration of the tongue. He inoculated 30 rats with human leprous tissue, with no other result than the production of granulomata, similar to those which are caused by the injection of almost any kind of foreign matter. Human leper serum is polyvalent, that is, it can deviate complement in the presence of divers antigens. Lepers react to various toxins, to mastin for instance, which is prepared from a streptothrix which is not *B. leprae*.

(ii.) This is the best monograph on leprosy which has been published lately. It embraces critical reviews of all recent work, to which a copious bibliography is given.

In the 13th century there were 19,000 leper houses scattered throughout Europe; 2,000 in France alone. In the 17th century the disease had almost disappeared. The infection still lingers in parts of Brittany, Provence, Alpes Maritimes and Auvergne. It is prevalent in Spain and Portugal, and in the countries along the Mediterranean. There is a small focus in Switzerland. Lepers were numerous in the Scandinavian peninsula, but their number is decreasing. It is estimated that there are 100,000 lepers in India, 13,000 in Indo-China, 25,000 in Japan, and 3,000 in the Philippines. Leprosy is the scourge of Oceania.

The incubation period is usually from 2 to 5 years, but it varies from 8 months to 32 years. Attacks of intermittent fever, dryness and hyperaesthesia of the skin, and rheumatic pains are among the first symptoms of the disease. Twenty references are given to papers on the serum diagnosis of leprosy. Many antigens have been employed with which positive results were obtained. In anaesthetic cases the serum is generally negative. The author doubts whether the *B. leprae* has been isolated, since no one has succeeded in inducing leprosy in the lower animals with cultures. Inoculation with leprosy material, however, is also unsuccessful. The lesions which follow the introduction of such matter into the eyes of rabbits are not due to the proliferation of the *B. leprae*, for the same changes occur when the bacilli are killed before injecting. Monkeys are immune. DANIELSSEN inoculated himself and nine of his pupils with leprosy discharges and tissues without exciting the disease. PROFETA and BARGILLI made similar unsuccessful experiments. Altogether about 60 people have submitted to such injections without harm. The criminal Keanu developed leprosy 6 months after the insertion of a leproma in his forearm. ASHMEAD states that in Japan vaccine lymph from leprosy infants is used with impunity. GAIRDNER, however (*Brit. Med. J.* 1887, Vol. 1, p. 1269), reported two cases of infection transmitted by vaccination (see also this *Bulletin*, Vol. 1, p. 192). The part played by contact is exemplified by the case reported by BENSON: a man who had never left Ireland contracted the malady by sleeping in the same bed and wearing the same clothes as his leprosy brother. Other similar instances are quoted. Several investigators have discovered leprosy bacilli in the droplets of mucus or saliva ejected by lepers. The author does not favour the view that leprosy is transmitted by insects. He states that acid-fast bacilli resembling the *B. leprae* are found frequently in many arthropods. Moreover the easily inoculable leprosy of rats is not conveyed by insects. The author dwells on the analogies between these two infections, and thinks that they bear the same relation to each other as the avian tubercle bacillus to the human. In both human and rat leprosy the bacilli may lie latent for long periods. Secondary infections can light up the disease in both instances.

The preventive measures in force in Norway have effected a reduction in the spread of leprosy. The leper lives with his family, but in a room apart, washing and boiling his own clothes and the articles used by him.

C. B.

BOECKMANN (Eduard). *Clinical Aspect of Leprosy.*—*Jl. Amer. Med. Assoc.* 1913. Sept. 20. Vol. 61. No. 12. pp. 946-949.

The initial lesion of leprosy, if it exists, has never been discovered. At the onset of the infection a change is noticed in the mental demeanour of the patient, he becomes listless, drowsy, and depressed, he complains of malaise and pains in his limbs; his face becomes puffy and of dusky hue, and his cornea is surrounded by a yellowish zone in his sclerotic.

In nodular leprosy the preliminary skin eruption consists of ill-defined livid or pigmented areas on the face and ears; the rest of the body remains free. In anaesthetic leprosy well-defined isolated erythematous spots appear on any part of the body except the scalp; these maculae may last for a few days only or they persist for months.

Lepromas usually remain stationary for years, after which they may undergo absorption or ulceration; healing may result. Eruptions of new nodules may occur suddenly; these outbreaks are accompanied with fever and constitutional symptoms.

In nerve leprosy the anaesthetic areas are generally found on the forearms and hands, on the legs and feet, and on the body, face and eyes. Acute rheumatic affections of the joints are not infrequent. As the neuritis proceeds, paresis and atrophy of muscles ensue ending in ulcerations and mutilations. The olfactory, optic and acoustic nerves escape.

C. B.

BREDA (Achille). *La Lepra del Globo Oculare e dei suoi annessi.*—*Gior. Ital. d. Malatt. Veneree e d. Pelle.* 1913. May 10. Vol. 54. (Anno 48.) No. 2. pp. 214-255.

The clinical histories of 37 lepers are given: 7 were suffering from nodular leprosy; 14 from anaesthetic; and 16 from mixed leprosy. Since the year 1890, 29 of these patients have developed leprous lesions of the eyes. Nastin, antileprol, salvarsan and neosalvarsan have had little or no curative effect. The thermocautery is useful in limiting the advance of the leproma towards the cornea.

Inoculations of leprous tissue into the cornea, anterior chamber, peritoneum, testis and skin of dogs, rabbits, and sheep failed to cause proliferation of the leprosy bacilli.

C. B.

DYER (Isadore). *The Dermatologic Aspects of Leprosy.*—*Jl. Amer. Med. Assoc.* 1913. Sept. 20. Vol. 61. No. 12. pp. 950-951.

Macular leprosy must be distinguished from syphilis and from erythema multiforme; from the former by the leprous eruptions

preferring the extensor surfaces, by their large size and by the dusky colour of the pigmented forms; from the latter by the duration of the leprous rash for weeks or months, and by the absence of fading on pressure in the pigmentary and infiltrated forms of macular leprosy. Iodide eruptions sometimes resemble leprosy.

The small tubercles of leprosy appear as clusters of pellets of bees-wax embedded in the skin of the back of the hands, chin, lips, and cheeks; they are anaesthetic. In lupus the tubercles are purple and ulcerate early, in syphilis the lesion is crescent-shaped, and leaves a pigmented scar. Xanthoma presents deep-seated, flat, smooth, lissom, yellow, tender tubercles.

Bullous leprosy is an early form which appears over the joints of the hands and feet; pigmented scars are left.

A diffuse pigmentation of the whole body may occur in leprosy; in vitiligo the margin is abrupt, in Addison's disease the pigment is localised, and in mycosis fungoides there is staining under furfuraceous scales.

In scaling leprosy there is wrinkling of the skin which becomes dirty gray and atrophic. In xerosis there is hypertrophy of the skin, and the desquamation is greater.

The neurotic leprous ulcer is recognised by its deep cylindrical cavity, its indurated well-defined edges, and its foetid odour.

C. B.

KIRBY-SMITH (J. L.). **Tubercular Leprosy in a Negress.**—*New York Med. Jl.* 1913. Oct. 11. Vol. 98. No. 15. p. 708. With 1 illustration.

The illustration shows extensive nodular leprosy of the face of a woman aged 22 years. *B. leprae* were found in the nasal ulcerations. The disease was of three years' duration and had not been benefited by chaulmoogra oil, but some improvement was observed after the exposure of the nodules to X-rays.

Jacksonville, Florida, where she had resided since infancy is almost exempt from leprosy; the source of infection could not be discovered; her parents and husband were healthy.

C. B.

DE BEURMANN & GOUGEROI. **Bacillurie et Bacillémie Hansénienne. Le Rein des Lépreux.**—*Lepre.* 1913. July. Vol. 14. No. 2. pp. 73-77.

A leper developed chronic nephritis accompanied with attacks of haematuria. After many examinations extending over several months, two acid-fast bacilli were found.

In another advanced case of leprosy acid-fast bacilli were discovered in the blood during a febrile attack, and also in the urine, although there were no signs of renal disease.

C. B.



BIEHLER (R.). *Wundheilung bei Lepra*. [Healing of Wounds in Leprosy.]—*Deut. Zeitschr. f. Chirurgie*. 1913. Sept. Vol. 124. No. 1/4. pp. 47-52. With 4 text-figs.

Lepers withstand surgical infection better than the healthy. Their wounds heal readily although sloughing. Cicatrization takes place more quickly in nodular than in mixed and nerve leprosy. The ulcers of pure nerve leprosy due to trophic changes contain no *B. leprae*; these are intractable.

Under nastin, antileprol, baths, tincture of iodine, oil of camphor, ichthyol and zinc perhydrol all the chronic ulcers on the legs and feet of a man who had been a leper for 8 years, healed in 3 months. *B. leprae* were abundant in the discharge. Two years later the lepromata had subsided, but the ulnar and peroneal nerves were affected; an ulcer broke out on his foot which resisted all treatment. Another leper of 5 years standing suffered from chronic ulcers of his legs which healed with the same remedies in 2 months. Nerve symptoms came on a year later and some time afterwards ulcers developed on the soles of both feet which were refractory to all remedies. The greater part of the left leg of a man who had been afflicted with leprosy for 16 years had been ulcerated for five years. Healing of the ulcer was complete in 7 months. Anaesthesia of the ulnar and peroneal nerves ensued; 4 months later the scar broke down and remained open permanently.

C. B.

BARBÉZIEUX (G.). *Contribution à l'Etude de l'Hérédité Lépreuse. Malformations congénitales observées chez Deux Consanguins issus de Parents Lépreux*.—*Rev. de Méd.* 1913. Sept. 10. Vol. 33. No. 9. pp. 737-744. With 4 text-figs.

Leprosy is not inherited, for the offspring of lepers removed from their parents remain free from the disease; but the progeny of lepers may be defective. The Cagots of the middle ages were the descendants of lepers inhabiting the Pyrenees, and though not all lepers themselves, most of them suffered from malformations of the hands or feet, or from some other deformity.

At Tonkin the daughter of leprous parents, aged 26, presented no signs of leprosy. Only a middle finger existed on each hand, the carpus was massed together, and all the metacarpal bones except the 3rd and 4th were deficient. The 2nd, 3rd and 4th metatarsal bones of both feet were wanting and there was fusion of two cuneiform bones with the cuboid.

The brother, aged 22, probably an early anaesthetic case, exhibited a nearly similar malformation in his hands and feet.

C. B.

#### MORBID ANATOMY.

SERRA (Alberto). *Di un Bazo Particolare Strutturale del Sistema Venoso Parenchimale nelle Capsule Surrenali di un Leproso*.—*Pathologica*. 1913. June 15. Vol. 5. No. 111. pp. 347-351.

Nodules of muscular tissue invaded the lumen of the veins of the medullary substance of the adrenals of a leper.

C. B.

LOMBARDO (C.). **Singolari Formazioni di Elastina entro Cellule Giganti in Casi di Lepra.**—*Giorn. Italiano d. Malattie Veneree e d. Pelle.* 1913. Mar. 28. Vol. 54. (Anno 48.) No. 1. pp. 75-79.

From one to six stellate bodies, 3-5  $\mu$  in diameter, which answered to the chemical and staining tests of elastic tissue were found in the giant cells of the skin lesions of two cases of leprosy. Similar inclusions have been seen by other observers in lupus, chronic inflammatory conditions of the lungs, and in uterine fibroids.

C. B.

#### TREATMENT.

HEISER (Victor G.). **Leprosy. A Note regarding the apparent Cure of Two Lepers in Manila.**—*U.S. Public Health Rep.* 1913. Sept. 5. Vol. 28. No. 36. pp. 1855-1856.

Leprosy bacilli were found in the scrapings of thickened reddish spots on the nose of a Filipino, aged 27, in the middle of the year 1909. Under vaccine [nature not stated] treatment for a year no change was noted. He was then given chaulmoogra oil, at first by mouth, then hypodermically combined with oil of camphor and resorcin for nearly a year. In June, 1913, he had apparently recovered; no *B. leprae* could be discovered, and he had been free from lesions for a year.

In January, 1910, scrapings of the red macules on the cheeks, forehead and chin of a Filipino woman, aged 22, contained leprosy bacilli. Injections of vaccine for 5 months, combined with the oral administration of chaulmoogra oil for 3 months, followed by chaulmoogra oil, camphor and resorcin hypodermically, resulted in an apparent cure. In June, 1913, there was no evidence of leprosy on clinical and microscopical examination.

There are now in the San Lazaro Leper Hospital numerous cases treated by chaulmoogra oil alone, which have been free from signs of leprosy for 22 months.

C. B.

RUDOLPH (Max). **Beitrag zur Nastinbehandlung der Lepra.** [Nastin Treatment of Leprosy.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Oct. Vol. 17. No. 19. pp. 669-671. With 1 plate.

The author has reported five cases of leprosy treated with nastin in which improvement occurred in all except one. He now records his sixth case. A leper who had been afflicted for 5 years and incapacitated for 2 years suffered from the mixed form of the infection, complicated with iritis. In the course of 18 months he received 3 injections of nastin B.<sup>0</sup>; 8 of nastin B.<sup>1</sup>; and 12 of nastin B.<sup>2</sup>. After 6 months' treatment the iritis disappeared and the anaesthesia was less; the lepromata on the hands and forearms were softer, but leprosy bacilli were still present. At the end of 18 months he had much improved, the

leonine expression of his face was much less marked, and *B. leprae* were not discovered in his nasal secretion. The photographs taken before and after treatment afford convincing evidence of the improvement which took place.

The author has not seen such favourable results with other remedies, including salvarsan and chaulmoogra oil, in his nine years' experience with hundreds of lepers. He advocates intermittent courses of nastin combined with chaulmoogra oil by the mouth.

C. B.

SERRA (Alberto). *L'Antiléprol dans le Traitement de la Lèpre.*—*Lepra*. 1913. July. Vol. 14. No. 2. pp. 63-69. With 8 figures.

This is a translation into French of the paper which appeared in *Giornale Italiano delle Malattie Veneree e della Pelle* (see this Bulletin, Vol. 2, p. 64).

C. B.

SERRA (Alberto). *Alcuni Casi di Siflide, Lepra e Psoriasi trattati col "606."*—*Giorn. Ital. d. Malattie Veneree e d. Pelle*. 1913. May 10. Vol. 54. (Anno 48.) No. 2. pp. 182-213.

A single intravenous or intramuscular injection of 0.4 to 0.6 gram. of salvarsan was given to three lepers without benefit. In all the complement deviation test with a leprosy antigen was positive.

C. B.

LOEHE (H.). *Beitrag zur Chirurgie der Nervenlepra*. [The Surgery of Nerve Leprosy.]—*Zum 4. Oktober 1913, dem 60 Geburtstag Seiner Exzellenz des Generalstabsarztes der Armee und Chefs des Sanitätskorps Prof. Dr. Otto v. Schjerning. (Eine wissenschaftliche Festgabe aus den Reihen des Sanitäts-offizierkorps.)* pp. 129-135. [1913. - Berlin: Ernst Siegfried Mittler und Sohn.]

Leprosy is a rare disease in Germany. Indigenous cases are limited to the extreme north-east of the Province of East Prussia.

A native of Brazil developed a spot on the back of his left hand in 1911, followed some months later by paralysis of the muscles supplied by the radial nerve which was thickened. It was therefore resected in January, 1913. A few *B. leprae* were found in it, although none were discovered in the nasal secretion or skin lesion. In discussing the diagnosis attention is drawn to the similarity of Boeck's multiple benign sarkoid of the skin, an infectious granuloma, to leprosy.

He quotes the case reported by CRAMER in which the whole of the median, ulnar, and external cutaneous nerves were excised. The patient recovered from leprosy and was in good health 18

years later. SEDERHOLM performed a similar operation on a leper who was free from active signs of the disease when examined nine years afterwards.

The author believes that anaesthetic leprosy can be cured by operation. [But see this *Bulletin*, Vol. 2, pp. 286 and 289, where it is noted that IMPEY states anaesthetic cases recover, and that SANDES aggravated the symptoms by excision of the nerve.]

C. B.

GOODHUE (E. S.). **The Surgical Cure of Leprosy, based on a New Theory of Infection.**—*New York Med. Jl.* 1913. Aug. 9. Vol. 98, No. 6. pp. 266-268. With 3 figs.

This paper is a confused medley of extracts from various writers, together with statements which are not in harmony with our knowledge of leprosy, such as "Leprosy is more frequently than otherwise primarily a localised disease; as such it may be eradicated by suitable topical remedies, or careful surgical interference," and "The whole period between infection and development of the primary lesion (incubation) may be as short as five days." No details are given.

C. B.

#### PROPHYLAXIS.

PEIPER (Otto). **Die Bekämpfung der Lepra in Deutsch-Ostafrika. Auf Grund amtlichen Materials bearbeitet.** [The Control of Leprosy in German East Africa.] — *Beihfte z. Arch. f. Schiff- u. Trop.-Hyg.* 1913. May. Vol. 17. Beiheft 4. 105 pp. [184-283.] With 34 text-figures, 3 plates, and 2 maps.

In the year 1909 there were nearly a thousand lepers segregated in leper villages and camps in German East Africa. In 1912 3,800 lepers and their families were living in 47 leper homes and villages. Besides these it is estimated that several thousand lepers were at large. The anaesthetic cases are twice as numerous as the nodular.

Of 31 lepers treated with nastin since the year 1907, three are believed to have recovered and six have much improved. Chaulmoogra oil had no good effect.

The administrative measures which have been taken for the suppression of leprosy in the 22 districts of the colony are described, and the details of the cost of maintenance of the leper community are given. It is good policy to decentralise rather than to collect the infected in one large asylum. The family life need not be disturbed, though there are risks. An instance is quoted of a woman in good health having lived with her leprous husband for many years; *B. leprae* were discovered in a small ulcer on her nasal septum.

C. B.

**BAYÓN (H.). Report by the Government Research Bacteriologist (Leprosy) on the Necessity or Advisability of Segregation in Relation to the Conditions and Spread of Leprosy in South Africa at the Present Time; the Measures to be provided for the Prevention and Cure of Leprosy; and the Suitability of Robben Island as a Place of Detention for Lepers.—*S. Africa Med. Rec.* 1913. May 24. Vol. 11. No. 10. pp. 187-194.**

In support of the author's opinion that lepers should be segregated, he quotes the case reported by de BEURMANN, in which a French sailor contracted leprosy after a stay of 20 hours only in an endemic area, and that recorded by LOCHTS where a leper infected 28 persons. He refers to GOLDSCHMIDT's account of a child who developed leprosy six years after being nourished for four weeks by a leprous wet nurse, and to SALTZMAN's history of a leper who infected four wives in succession. He states that there are two observations which show that the disease was contracted in the United Kingdom from cases imported from abroad. In Russia and the Dutch East Indies, where restrictive legislation has been allowed to lapse, the number of lepers has increased. The adult healthy relatives of lepers may live in the settlement if they conform with the regulations, since lepers under proper supervision generally cease to be a source of infection.

The three leper asylums and the two leper settlements in the South African Union can accommodate less than two-thirds of the total leprous population. The water supply of Robben Island is defective though otherwise the island is not unsuitable for a leper settlement, if the other residents were removed.

C. B.

**GOMES (E.) & TERRA (F.). Prophylaxia da Lepra.—*Brazil Medico.* 1913. July 22. Vol. 27. No. 28. pp. 288-289.**

The establishment of colonies of lepers is advocated, in which those that are able shall have facilities afforded them of useful employment. There should be a small hospital which must contain an operation theatre and a laboratory. An incinerator is necessary.

C. B.

**VAMPRÉ (Enjolras). Prophylaxia da Lepra.—*Revista Med. de S. Paulo.* 1913. Feb. 28. Vol. 16. No. 4. pp. 76-78.**

It is urged that leper colonies and asylums should be established in the State of São Paulo where leprosy is on the increase; in 14 towns alone there are 300 lepers. The benefit of prophylactic measures is shown by the experience of Norway, where there were nearly 3,000 lepers in the year 1856; in 1907 their number had been reduced to 438. Japan has spent 900,000 marks on the construction of leper sanatoria; in the Sandwich Islands a quarter of a million sterling has been devoted to the same purpose.

The author gives a draft of bye-laws which he thinks would be effective in controlling the spread of leprosy in the State of S. Paulo.

C. B.

## RAT LEPROSY.

**MEYER (Carlos).** Instituto Bacteriologico do Estado de S. Paulo. **Relatorio sobre a administracao e os trabalhos do Instituto durante o anno de 1912.** [Report of the Bacteriological Institute of S. Paulo for the year 1912.]—*Revista Med. de S. Paulo*. 1913. Feb. 15. Vol. 16. No. 3. pp. 56-61. [Leprosy of Rats. p. 60.]

Among 135 rats sent to the laboratory under suspicion of plague infection rat-leprosy was discovered in two. Acid-fast bacilli were seen in the enlarged lymphatic glands. It was not known previously that the disease existed in Brazil, or other parts of South America.

C. B.

## PLAGUE.

LISTON (W. Glen). **Plague.**—*Trans. xvii. Intern. Congress of Med. London, 1913.* Section xxi. Trop. Med. & Hyg. Part 1. pp. 9-23: and [in abbreviated form] *Jl. Trop. Med. & Hyg.* 1913. Sept. 15. Vol. 16. No. 18. pp. 273-275.

The paper is in two sections. The first deals with "The Epidemiological Features of Bubonic and Pneumonic Plague contrasted."

Plague pneumonia epidemics in India have not been infrequent. They have however always been limited in size in response to prompt measures. In the case of the bubonic form of the disease, however, the infection was more difficult to control and the infective property appeared rather to belong to the home than the patient. This led many workers to search for the bacillus in infected houses, without any success. The connection between rats and plague and the suggestion that the rat flea was an infecting agent led Liston to use guinea-pigs as flea traps, and in the stomachs of fleas so captured in infected houses he was able to demonstrate the plague bacillus. He showed, moreover, that rat fleas could be caught on men and this suggested an obvious mode of infection. Laboratory work by French workers showed plague was conveyed from rat to rat by the flea. The Indian plague commission showed infected rats were not directly infectious in the absence of fleas.

The international commission appointed by the Chinese Government showed that in the case of the pneumonic type the rat played no part in its spread. Man to man infection was the rule and though many plague houses were rat-infested the rats did not take the disease. It was shown that the bacillus of pneumonic plague was no more virulent than the variety obtained from bubonic cases.

STRONG has shown that the spread of infection in this variety is largely due to the coughing of the patient, and the contaminated area varies in radius according to several factors. Liston is of opinion that one of the important factors is air temperature and that the radius of spread of the droplets will be greater in colder atmospheres. This he believes is the reason of pneumonic plague prevailing in cold and temperate climates more than in hot ones. Some historical corroboration of this view is given. The pneumonic plague was easily checked in China on the adoption of suitable measures, but in India on the other hand bubonic plague does not yield to the most strenuous hygienic efforts of the administration.

Part II. Liston points out the long intervals between epidemics and that these epidemics depend on rat epizootics. These two facts he endeavours to correlate with the different susceptibility of the rat in different districts at different times. He shows that Madras rats (*Mus rattus*) are much more susceptible to plague than Bombay or Poona rats, tested by the subcutaneous injection of known quantities of bacteria or by the method of natural flea infection. Now Madras has been practically free from plague, while the other cities have been severely attacked by it, so that we have to believe that the existence of plague in a centre is

associated with the production of a relatively immune race of rats and this immunity ultimately reduces the possibility of the natural epizootic. This immunity of the rat population is transmissible to their young.

W. J. Penfold.

**KITASATO (S.). i. On the Value of the Search for Rat Fleas in the Detection of Plague.**—*Trans. xvii. Intern. Congress of Med. London. 1913. Section xxi. Trop. Med. & Hyg. Part 1. pp. 1-7.*

ii. **Ueber die Pest. Die Wichtigkeit des "Rattenflohes" zur Feststellung der Verbreitung von Pest.** (Nach einem Vortrage, gehalten in dem xvii Internationalen medizinischen Kongress, London, 1913).—*Berlin. Klin. Wochenschr.* 1913. Oct. 13. Vol. 50. No. 41. pp. 1881-1884.

The papers contain a résumé of observations made at Kobe in 1909 and 1910.

The plague incidence in rats and men was found maximal in winter. From trapped and purchased rats 3,234 fleas were examined. *Ceratophyllus* constituted 86·3 per cent. while the common Indian rat flea formed only 4·7 per cent. All the types except *X. cheopis* were most prevalent in the winter during the months January to June, while *X. cheopis* was most prevalent in November and had disappeared by March. Following the recommendations of the Indian Plague Commission guinea-pigs were allowed to run freely for 24 hours in suspected houses; the fleas so obtained were examined in reference to plague infection and of course the plague mortality of the guinea-pigs was noted. The guinea-pig mortality from the sojourn in plague and neighbouring houses was but slight and about one-sixth of the fleas from the same houses was found infected.

A comparison of the degree of infectivity of houses in which plague rats had been found and other houses near resulted in showing the latter to be more infected. Kitasato explains this by stating that rats rove a good deal and of this he gives a striking illustration.

He shows how useless ordinary disinfection of houses is in getting rid of fleas and the great value of rat proofing for the purpose. His figures bearing on this point are very striking.

He believes the plague germ may be best detected by the guinea-pig test and this constitutes one of the most important factors in plague prevention.

W. J. P.

**WU LIEN TEH [G. L. TUCK]. Investigations into the Relationship of the Tarbagan (Mongolian Marmot) to Plague.\*—*Lancet.* 1913. Aug. 23. pp. 529-535. With 4 text-figs; and *Jl. Trop. Med. & Hyg.* 1913. Sept. 15. Vol. 16. No. 18. pp. 275-280.**

The foundation of the Manchurian Plague Prevention Service as a result of the recommendation of the International Plague

\* Abstract of paper read at the International Congress of Medicine, 1913, Section of Tropical Medicine and Hygiene.



Commission is briefly described. Many of the delegates of that commission believed that the marmot was closely connected with the spread of plague. As a result of this belief the author organized an expedition into the regions where marmots abound, to see what justification could be found there for the view. He describes the itinerary of this expedition. The Chinese expedition worked for some little time in conjunction with a Russian expedition under ZABOLOTNY. The centres of work were Manchouli in Manchuria, Borsja in Siberia, and Charhada and other places in Mongolia. The housing of the hunters is dealt with at full length and is shown to be as bad as possible from the hygienic aspect. Rumours from various localities of mortality amongst tarbagans were acted upon in directing the search, but in no case was a plague infected animal (living or dead) found. Apart from one such naturally infected animal described by ZABOLOTNY there seems to be no definite evidence of plague naturally occurring in the tarbagan. A description of the tarbagan (*Arctomys bobac*) is given. The author found on the captured tarbagans fleas and ticks; all the fleas were of one type (*Ceratophyllus silvaticus*) and these were shown to bite the human subject, though they were not apparently very anxious to do so.

The author concludes that the direct relationship of the tarbagan to human plague may be looked upon as negligible.

W. J. P.

BULLETIN DE L'OFFICE INTERNATIONAL D'HYGIÈNE PUBLIQUE.  
1913. Sept. Vol. 5. No. 9. pp. 1544-1551.—**Prophylaxie de la Peste dans le Gouvernement d'Astrakhan (Russie).**  
(Communication de la Commission Impériale pour la Lutte contre la Peste.)

In 1877 an epidemic of plague appeared in Astrakhan. From that date till 1899 the district remained free, but from 1899 to this year plague has appeared each year in the same tribe of Boukeiv. According to careful enquiries it appears that the epidemics are due to some local latent nucleus of infection.

Although the causation and mode of propagation still remain doubtful it appears probable that the hamster plays a great part in the dissemination of the disease in this region.

Plague infection of the camel was proved to occur and also individuals who had eaten the flesh of infected camels died of the disease. In consequence of these facts a commission was appointed to examine the part played by the camel in the spread of the disease; out of 15,000 examined not a single infected animal was found.

The government of Astrakhan, recognising the part played by the hamster, suggested a government grant for destroying this pest but in this they were opposed by the council of the empire who held that the role played by the hamster was not sufficiently

demonstrated to warrant this; in consequence a special commission of the Imperial Institute of Experimental Medicine has been charged to solve this problem.

W. J. P.

LURZ (Richard). *Eine Pestepidemie am Kilimandscharo im Jahre 1912.* [A Plague Epidemic on the Kilimanjaro Mountain, German East Africa.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Sept. Vol. 17. No. 17. pp. 593-599.

A general description of the administrative arrangements of the district and nature of the inhabitants is followed by a few historical notes on plague as locally observed. Some doubt appears to exist as to whether the plague raged in 1898 in the part of the colony under discussion. In March, 1912, however, plague broke out and was identified by bacteriological as well as clinical investigation.

In Gasseni 55 natives died of pneumonic plague between 15th March and 14th April and three died of bubonic plague in the same time. In Usseri Kwai Demassi eleven died of pneumonic plague about the same time. Incubation period was one to three days, the disease lasted one day as a rule and the mortality was 100 per cent.

The usual methods of control and burning of the plague infected huts were adopted as control measures. Very few rats were present in the infected region and no marked mortality was observed amongst them. The type of rat found in the huts was almost exclusively *Pelomys fallax iredescens* (Heller). Around the huts many tree rats were found; field rats were also common. The latter were never found infected with plague though they are susceptible to it.

The fleas found on the men, rats, and dogs are described. *Locmopsylla cheapis* and *Sarcopsylla gallinacea* being the most frequent. Of nearly 3,000 house rats examined 36 were found plague infected while three infected tree rats were found in an examination of 3,297. Over 3,000 house rats in the southern portions of the colony were examined and no plague infected animal found amongst them.

Rat and mouse destruction was carried out very thoroughly, over 30,000 being killed in nine weeks. The holes of rats were dug out in their entire length so that but few rats of infected houses could have escaped. Attempts to use guinea-pigs as traps for infected rat fleas were not successful in a house where a case of plague-pneumonia had died.

W. J. P.

MEYER (Carlos). *Instituto Bacteriologico do Estado de S. Paulo. Relatório sobre a Administracao e os Trabalhos do Instituto durante o Anno de 1912.*—*Revista Med. de S. Paulo.* 1913. Feb. 15. Vol. 16. No. 3. pp. 56-61. [Bubonic plague, p. 58.]

During the year 1912, 135 rats were examined in the capital for plague, of which 12 were found to be infected. Meyer suggests that large numbers should be examined daily instead of small irregular numbers as heretofore.

Seven samples of lymph from glands of suspected plague cases gave one positive result. The bacillus was isolated and identified.

Suspected material examined showed three positive cases in Quiririm, three in Redempecao and eight in Taubaté.

Plague pneumonia was suspected in a case in the isolation hospital of the capital but the examination of sputum was negative.

The staff of the institute made autopsies in five cases of plague; cultures were obtained in each case.

W. J. P.

**MONTEL.** *Lésions Bucco-Pharyngées dans la Peste.* [Clinique d'Outre-Mer.]—*Ann. d'Hyg. et Méd. Colon.* 1913. July-Aug.-Sept. Vol. 16. No. 3. pp. 779-781.

This short note gives an account of five patients who suffered from plague at Saigon in 1908. These patients all showed cervical adenitis as a result of lesions of the mouth and pharynx; the lesions were principally tonsillar and clinically similar to diphtheria. Montel believes the tonsils constituted the point of entrance of the infection.

W. J. P.

**PIRAS (L.).** *Die Präzipitinreaktion als diagnostisches Mittel der Pest.* [The Precipitin Reaction in the Diagnosis of Plague.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Sept. 27. Vol. 71. No. 1. pp. 69-80. With 1 text-fig.

It has been shown by KISTER and SHUMACHER that dead plague-infected rats kept at a temperature of 20° C. are liable after seven days to give a negative result if an endeavour is made to prove by culture or animal inoculation that they are infected. GRYZEZ and WAGON stated on the other hand that extracts of old plague-infected tissues specifically deviate complement in the presence of an anti-plague serum.

Piras has controlled these last results. He used an extract in carbolized saline. He found that the specific deviation of complement only took place up to the sixth day after death and was consequently no more useful than the "Inoculation of Animals" test. He then attempted to get positive results by the precipitin test. His preliminary experiments against a good serum showed that the precipitinogen was present in highest concentration in the liver and spleen and for his further experiments he confined his attention to mixed extracts of these organs.

His work shows that a specific precipitinogen is present in the cadavers of experimentally plague-infected rats and guinea-pigs even when very advanced putrefaction has set in and this specific body can be easily identified even up to 68 days after death. The same body is present in the faeces of plague infected animals. This method can be used after all other methods have become quite useless owing to advancing decomposition.

Piras obtained constant results using sera from different outside sources and in addition sera made by himself. The fact that

the precipitinogen is present in faeces may enable one to determine the presence of disease in rats though no infected rats have been caught.

W. J. P.

#### RATS IN RELATION TO PLAGUE.

ROBINSON (George H.). **The Rats of Providence and their Parasites.**—*Amer. Jl. of Public Health.* 1913. Aug. Vol. 3. No. 8. pp. 773-776.

From July, 1912, to January, 1913, 341 rats were examined; 333 of these were the brown rat (*Mus norvegicus*). The sizes varied from 7 to 18 inches and 59 per cent. of the rats were adults. 195 of the rats harboured amongst them 2,053 fleas, *i.e.*, 11 per infested rat. One rat had no less than 300. 75 per cent. of the fleas were *Xenopsylla cheopis* and 22 per cent. *Ceratophyllus fasciatus*.

The fleas were found fairly equally distributed over the rats, whereas in Australia they were mainly found on the head and neck regions and in San Francisco they were most prevalent on the hinder quarters of the animals. In cold weather the fleas tend to accumulate on the more thickly furred portions of the body. The total number of fleas present was markedly reduced in the cold weather.

21 per cent. of the rats were infested with mites. The common rat louse was found on 24 per cent. of the animals.

The liver in 7 per cent. of the rats showed the encysted form of the cat tapeworm *Taenia crassicollis*. These were especially frequent in rats taken from the local meat markets.

W. J. P.

HEISER (Victor G.). **The Rats of our Cities. What becomes of the Carcasses of Rats dying Natural Deaths?**—*U.S. Public Health Reports.* 1913. July 25. Vol. 28. No. 30. pp. 1553-1554.

This short paper takes the form of a conundrum. It is pointed out that the rat census of a city is usually supposed to equal the human, further that the average life of a rat is five years. Now if only one rat be allowed for every two inhabitants in Manila there must be an average daily mortality of 82 rats. Refuse disposal in Manila is practically confined to that which occurs at the public crematory, as stoves in houses are unknown there and fires are most primitive owing to the high price of fuel. A sanitary officer was stationed at the crematory for one month and only discovered in the refuse one dead rat out of the 2,500 that died during the month. Heiser pertinently asks what became of the rest. If they were eaten by their fellows portions of carcasses and skeletons would be found in sanitary work but they are not. They don't die in the drains as they are a closed system. Gangs of men employed in killing rats practically never find dead ones unless poison has been laid. This puzzle is not elucidated by the author.

W. J. P.

FOR (F. Arthur). **Destruction of Rats in the Port of Rangoon.**—*Brit. Med. Jl.* 1913. Aug. 23. pp. 439-441.

The author first enumerates the occasions calling for the destruction of rats in vessels in plague prophylaxis. These are (1) When the vessel is plague infected. The plague infection may be a human case. The occasional difficulties of justifying rat destruction from this cause are dealt with. The case may not be sufficiently advanced to admit of exact diagnosis, or the patient may be reported as suffering from plague after having left the ship, the reloading of which may have already commenced; moreover the human case may not have infected the rat population of the vessel. If the plague infection be of rats as indicated by definite rat mortality it is obviously very necessary to destroy the rats. No such cases have been reported by commanders of vessels entering the port. The author suggests that routine trapping and bacteriological examination of rats on vessels would be a valuable index to the need for rat destruction.

(2) Rat destruction is discussed as a routine procedure, when the vessel is undergoing annual repairs and refits. In Rangoon this does not occur to any great extent as Rangoon is not the headquarters of many of the shipping companies.

(3) Routine rat destruction when a vessel is going to a port which makes this a condition of admission to the port.

Different methods of rat destruction are discussed and a number of experiments recorded which however were frequently interrupted from various causes or vitiated by factors beyond the control of the experimenter. In sulphur dioxide fumigation the actual percentage of gas used varied between  $1\frac{1}{2}$  and 6 per cent. and rat destruction occurred with percentages as low as  $1\frac{1}{2}$  per cent. The plague bacillus was actually found only once amongst the rats obtained from 22 "deratizations" of vessels with the Clayton apparatus. Tabular statements of the numbers of rats and mice found in different vessels, and their distribution in the vessels are given.

W. J. P.

DALEY (W. Allen). **Rats and their Extermination.**—*Public Health.* 1913. Oct. Vol. 27. No. 1. pp. 23-28:

The paper is an account of the present position of our knowledge on the subject. There are few new facts. The different types of rat which play a part in plague transmission are described and their geographical distribution considered. The types and prevalence of rat fleas are also described. A section dealing with rate of increase of rats and the economic damage they cause is also given. The occurrence of rat plague without human plague is discussed and shown to be due to the fact that *Mus decumanus* shuns man whenever possible, while on the other hand *Mus rattus* is closely associated with man. Illustrative cases of this point are given.

The diagnosis of rat plague is next discussed and the points observed in post-mortems of infected rats reviewed. The Rat Laws of several countries are passed in review and also the various methods of rat destruction.

W. J. P.

STRICKLAND (C.). The Bionomics of the Rat-Flea. [Correspondence.]—*Brit. Med. Jl.* 1913. Aug. 16. p. 435.

This letter deals with the question of the longevity of the fasting rat-flea. Strickland from personal observation came to the conclusion that adult fleas could live in hygroscopic rubbish for eighteen months. BACOT's criticism of this view was unfavourable and he attributed the facts observed by Strickland to long larval rests within the cocoon.

In this letter Strickland returns to the question. He believes BACOT has no evidence of value showing that fleas are unable to live a great length of time in the conditions of his experiments. Further BACOT has no evidence of larvae living in the resting stage eighteen months, his periods of observation never extending over 400 days. Strickland was also able to show *Trypanosoma lewisi* in several of his fleas which had fed on an infected rat eighteen months previously. It appears more experimental research is needed on the point.

W. J. P.

## MYIASIS.

FRANCAVIGLIA (M. C.). **Altro Caso di Myiasis nell'Uomo per Larva cuticolare d'*Hypoderma bovis* (De Geer).** [Another Case of Human Myiasis due to the Larva of *Hypoderma bovis*.]—*Policlinico*. Sez. pratica. 1912. Oct. 27. Vol. 19. No. 44. pp. 1593-1595.

Three cases which have occurred in Catania are referred to, one described in 1878 by BERRETTA, another in 1904 by the author, and the present one. The patient was a peasant child, fourteen years of age. In the beginning of October, 1911, a radiating pain began in the region of the umbilicus. After purgatives this decreased in intensity, but quickly returned, being more severe then than at first and now accompanied by fever. About the same time pain appeared in the right parietal region and a swelling about the size of a hazel nut developed from which a small white larva eventually escaped. Unfortunately this was not kept. About a month later a similar swelling appeared over the sternum and another larva was recovered. This on examination proved to be the young stage of the fly *Hypoderma bovis* (de Geer). Nothing appeared at the site of the original pain in the abdomen and the author thinks it may only have been due to some dietetic error.

[Dermal myiasis due to the *Hypoderma bovis* are common in cattle. Though rare in man cases have been reported from time to time. The eggs of the fly after being laid on the skin of the animal are licked off by the tongue and so find their way to the mouth and alimentary canal. From here the larvae are supposed to bore their way through the oesophagus and finally come to rest under the skin forming tumours. Escaping by ulceration they fall on the ground, become pupae, and so develop into the adult fly.]

G. C. Low.

BALZER (F.), DANTIN, & LANDESMANN. **Un Cas de Myiase rampante due à l'*Hypoderma bovis*.**—*Bull. Soc. Française de Dermatol. et de Syphiligraphie*. 1913. April. Vol. 24. No. 4. pp. 219-226. With 3 figs.

The case was that of a young girl born in Brittany who was employed in looking after cattle which had come for the most part from Scotland, Denmark and Norway. Later she came to Paris where a diffuse swelling developed above the right ear. This was accompanied with severe pain. Some days later the pain became localised to the inferior right temporal region just above the ear, and next a small orifice appeared from which the extremity of a mobile larva could be seen protruding.

Some days later the patient began to suffer from swelling and pain above the left shoulder. From this site another larva was recovered. Finally a third specimen appeared in the region of the right anterior superior iliac spine. BRUMPT to whom the larvae were shown identified them as those of *Hypoderma bovis*. The authors remark that French literature is not rich in cases of

this nature. Two cases, occurring also in Brittany, have however been observed and published by TOPSENT. In the first instance, a young girl of eight years, who lived in proximity to cattle, was attacked. The second case observed was also in a girl, six years of age, who lived on a farm with her parents. In the work of DUBREUILH the authors have found five cases mentioned, these having been observed in Belgium, Italy and Germany. Three of these were cases of infection with *Hypoderma bovis*, two with *Hypoderma diana*. Other cases have been observed by MAC'ALMAN, and WALKER in the Shetland Islands and by MURRAY in Ireland.

[FRANCAVIGLIA's paper (see above) furnishes further examples.]

G. C. L.

RUDELL (G. L.). **Creeping Eruption. Two Cases with Recovery of the Larvae.**—*Jl. Amer. Med. Assoc.* 1913. July 26. Vol. 61. No. 4. p. 247. With 3 figs.

Creeping eruption, according to the author, is a very rare disease and the recovery of the larva producing this curious lesion is rarer; only two reports are to hand of their discovery. The author records two cases seen by himself, particularly because in one of them, in which there were two of the lesions, both larvae were recovered. One was found 2 mm. from the inflamed end of the line, its position being indicated by a small dark spot. Lifted out and placed under the microscope its movements, which were limited to a maggot like wriggling, could be studied. The second specimen was recovered in a similar way to the first. A photograph of the boy with the line like lesions on his face is given, also a microphotograph of the larva found in the case, together with SOKOLOV's drawing from PUSEY's Dermatology of the larva of creeping eruption. [The author does not seem to have had his larvae identified. This would be interesting, and as he has them mounted in glycerin, is still possible.]

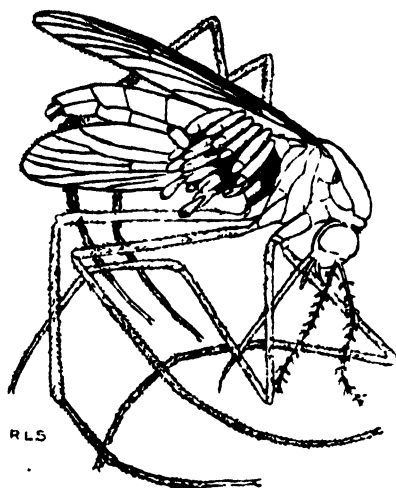
G. C. L.

SURCOUF (Jacques). **La Transmission du Ver Macaque par un Moustique.**—*Compt. Rend. Acad. Sci.* 1913. May 5. Vol. 156. No. 18. pp. 1406-1408. With 2 text-figs.

It is pointed out that in spite of the frequency of Ver Macaque tumours, we are still ignorant of the method in which these parasites, the larvae of *Dermatobia cyaniventris*, reach their host. In 1900 BLANCHARD observed the presence of packets of eggs attached to the abdomens of mosquitoes from Central America. MORALES of Costa Rica next believed that the *Dermatobia* laid its eggs directly on the abdomen of mosquitoes and that the larvae were transmitted to vertebrates when the mosquitoes sucked their blood. This assertion would explain the term *Gusano de Zancudo* and *Ver de Moustique* [Mosquito Worm of British Guiana] given by the Venezuelans to the Ver Macaque. The author points out however, that it seems incredible that the *Dermatobia*, which is as large as a blue-bottle fly, could lay its eggs directly on the



mosquito. RINCONES of Caracas believes that the eggs are laid on damp leaves in wet damp places frequented by the *Janthinosoma lutzi*, the mosquito which would appear to act as the carrier.



*Janthinosoma lutzi* ♀ carrying eggs of *Dermatobia cyaniventris* Macquart ( $\times 6\frac{1}{2}$ ).

The packets of eggs are enclosed in a strongly adherent cement substance which becomes softened and viscid when placed in water, and by this means they are supposed to become attached to the wall of the mosquito's thorax [Vide diagram.] There they ripen and opening give birth to a larva which wriggles in under the skin by way of the puncture made by the mosquito. TOVAR of Maturin, Venezuela, observed such mosquitoes carrying these eggs and by placing them upon animals obtained furunculous tumours. These, when excised in eleven days, showed in each a typical mosquito worm, from which ultimately emerged a typical adult *Dermatobia cyaniventris* fly.

[This is a very interesting note and it would seem from it that the puzzle of the passage of the larva of the *Dermatobia* to man and other animals had been solved.]

G. C. L.

**ZEPEDA (Pedro). Nouvelle Note concernant les Moustiques qui propagent les Larves de *Dermatobia Cyaniventris* et de *Chrysosomia Mucellaria* et peut-être celle de Lund, et de la *Cordilobia Anthropophaga*. — Rev. de Méd. et d'Hyg. Trop. 1913. Vol. 10. No. 2. pp. 93-95.**

The inhabitants of the Atlantic coast of Nicaragua talk with terror of a mysterious mosquito, which inoculates by its puncture a larva of variable dimensions and form. The puncture is not noted for the first few hours but after this, pain, redness and oedema appear. After 24 hours a furunculous tumour of a red colour and a hard consistence develops. Incision of this reveals the presence of the larva of a fly.

The author in studying the question observed on some mosquitoes [varieties not stated] white masses disseminated on the femur and on the pro-thorax and antennae. The bites of such insects produced the characteristic tumours with symptoms more or less severe, inflammation and suppuration. In the case of a young man of 24 years, of good constitution, bitten on the left forearm, violent inflammation occurred ten hours afterwards. The day after that the pain was intolerable though the tumour had not developed much in size. The temperature was raised to 100° F. The author then intervened and extracted the larva, applying antiseptic dressing to the wound. Cicatrisation quickly occurred and all the symptoms disappeared. In another case the larva developed but with much less acute symptoms. It came out in seven days and after this healing was rapid. An examination of the mosquitoes demonstrated to the author that the white masses or protuberances were the larvae and eggs of *Dermatobia cyaniventris*.

Further experiments gave the same results, but in one of these the author was surprised to see two perforations in the tumour from which a whitish liquid exuded. The larva extracted was different in appearance to those seen before and was finally identified at Philadelphia as that of the *Chrysomya macellaria*. [This is specially interesting.]

As to how the eggs and larvae of the fly reach the mosquito, the author believes that they become attached to it when it settles on rotting bananas and other putrefactive material.

[It will thus be seen that Zepeda's observations bear out those of SURCOUF *vide supra*.]

G. C. L.

NEIVA (Arthur) & GOMES DE FARIA. **Myiasis humana, verursacht durch Larven von *Sarcophaga pyrophila* n. sp.** [Human Myiasis, caused by Larvae of *S. pyrophila*.]—*Mém. Inst. Oswaldo Cruz*. 1913. Vol. 5. No. 1. pp. 16-22.

The case occurred in a young girl, ten years of age, who had sustained contusion on the right parietal region. The wound suppurated and in the pus fly larvae were ultimately found. The authors believe that the species is a new one and described it under the name of the *Sarcophaga pyrophila*, n. sp. The type has been preserved in the Oswaldo Cruz Institute. It is pointed out that the most common forms of myiasis in Brazil are those due to *Chrysomya macellaria* and *Dermatobia noxialis* [= *cyani-ventris*]. The first infection is known locally as *bicheira* or *bicheiro*, the latter as *berne* or *ura*. The following *Sarcophaga* have been found in human myiasis:—*S. carnaria*, *magnifica* and *latifrons* in Europe, *ruficornis* in India, *lambens* in Brazil, a sp. (?) in British Guiana by DANIELS and now the new species *pyrophila* described above.

[The *Sarcophagidae* are the common blow-flies, *S. carnaria* being the one most usually met with in Europe. Attracted by

the foul smell of ulcers or other lesions in the human subject the flies sometimes deposit their eggs on such sites and these developing into larvae give rise to this form of myiasis.]

G. C. L.

DE MOURA (Cursino). **Myiase do Seio.**—*Revista Med. de S. Paulo*. 1913. Jan. 15. Vol. 16. No. 1. p. 1.

A case of myiasis of the breast in a patient who suffered from an ulcerating cancer affecting both of these organs. The larvae, according to the author, were those of a fly locally known as "Mosca varejeira" = *Musca putrida* L. The presence of the larvae caused a further destruction of the tissues of the already diseased breasts.

G. C. L.

HECKENROTH (F.) & BLANCHARD (M.). **Note sur la Présence et l'Endémicité d'une Myiase furonculaire au Congo français.**—*Bull. Soc. Path. Exot.* 1913. May. Vol. 6. No. 5. pp. 350-351.

Observations made by one of the authors in different regions of the Congo, over many years, have enabled him to establish the rarity of the ver du Cayer [*Cordylobia anthropophaga* E. Blanchard] in these parts. Two cases of furuncular myiasis were, however, met with in the regions of Sangha and n'Goko, one of these in 1907, at Nola, in a European who had two larvae in his left arm, the other at Brazzaville. These were both attributed at the time to the *Cordylobia anthropophaga*, but the authors now think that they may really have been due to *C. rodhaini* described by GEDOELST. This species exists all over the Belgian Congo, notably at Leopoldville, but so far has not been described in the French Congo. A dog belonging to one of the patients was also affected.

G. C. L.

RODHAIN (J.), PONS (C.), VANDENBRANDEN (F.), & BEQUAERT (J.). **Rapport sur les Travaux de la Mission Scientifique du Katanga, Oct. 1910 à Sept. 1912.** pp. 171-186. (Observations sur la Dispersion Géographique et la Biologie du "Ver de Case" (*Auchmeromyia luteola* F.) et du "Ver du Cayer" (*Cordylobia Anthropophaga* Grünb.))—1913. Brussels: Hayez, Imprimeur de l'Académie Royale. [Royaume de Belgique—Ministère des Colonies.]

Observations made upon the *Auchmeromyia luteola* (Ver de Case) have demonstrated that this fly is oviparous. From the egg which is laid on damp soil a larva eventually emerges [the Congo floor maggot]. This creature is very active, disliking the light and rapidly escaping from it into the dust or earth. In the soil of different villages numerous larvae at different stages and pupae were found. The geographical distribution of the fly is a wide one, many parts of the Congo harbouring it.

The means of entrance of the larva of the *Cordylobia anthropophaga* under the human skin were demonstrated. At the moment of the exclusion of the egg from the fly one of the poles opens irregularly and a very small mobile larva emerges.

One of the authors placed such a larva on the skin of his arm and the following points were noted:—Without displacing itself the parasite sought immediately to penetrate through the intact skin. It attacked this with its buccal hooks and rapidly buried itself in the thickness of the epidermis. In three and a half hours it was already half way through the skin, penetrating this obliquely and producing a slight swelling. In six hours it had passed completely through. The penetration was accompanied by a very slight pricking sensation and itching. After this it was only with difficulty that the orifice with the posterior extremity of the larva was seen.

Unfortunately in this individual instance pressure was made on the spot and the larva came out of the skin and, though placed again on it, did not penetrate but died. Similar experiments on monkeys and dogs resulted in infection.

The authors however believe that the fly only rarely lays its eggs direct on man or other animals. In the majority of cases it would seem to deposit them on the ground where there is an animal or human odour. This would explain the localisation of the tumours, which are so often found in parts of the skin which have come in immediate contact with the soil and also the fact of the larva never being encountered on the backs of animals. Two cases of infection in Europeans are described. No cases of myiasis due to the *Cordylobia rodhaini* (Lund's larva) were noted.

G. C. L.

SERGEANT (Edm. & Et.). **La "Tamné," Myiase Humaine des Montagnes Sahariennes Touareg, identique à la "Thimni" des Kabyles, due à *Oestrus ovis*.**—*Bull. Soc. Path. Exot.* 1913. July. Vol. 6. No. 7. pp. 487-488.

The authors call attention to the fact that in 1907 they showed that the *Oestrus* of the sheep was frequently met with in the conjunctiva and nasal cavity of man in certain elevated regions of Kabylie, where the number of sheep as compared to the number of people was small. The presence of these larvae produces very disagreeable symptoms.

The Kabyles call the fly *Thimni*. In the Central Sahara in the Ahaggar mountains the people are also much annoyed in spring by flies which deposit their eggs in the conjunctiva and nasal mucosa and the name given to the fly there is *Tamné*. These names the authors show are synonymous and both apply really to the *Oestrus ovis* Lin. 1761.

G. C. L.

EDGAR (C. L.). **A Case of Screw-Worm in the Nose.**—*Texas State Jl. of Med.* 1913. May. Vol. 9. No. 1. p. 21.

Details of the case are given. Clinically there was a bloody, watery discharge from the nose which had a very offensive odour.

Many maggots were present. Treatment was by chloroform inhalations, these proving efficacious. The patient then returned home but later developed severe nasal haemorrhages from which he died. The larvae were those of the *Chrysomya macellaria* Fab. 1794.

In Texas, according to the author, the life of this fly extends from June until autumn. The female is oviparous and lays from 200 to 400 eggs at one laying. These hatch in about nine hours and the larvae develop very rapidly. The female fly when ready to deposit her eggs is attracted by any foul odour, such as a foul smelling discharge from the nose or other situation, and does not require to remain very long in any one particular place in order to deposit her eggs.

In the wound the larvae are found burrowing for from four to six days, after which they leave this site and crawl into the earth, there transforming into the quiescent pupal stage. After this stage has lasted for one or two weeks the mature fly appears. From two to three weeks are therefore required for the entire life cycle.

G. C. L.

FIELD (F. E.). **Myiasis: With Special Reference to some Varieties Treated at the Georgetown Hospital.**—*Brit. Guiana Med. Ann.* for 1911. pp. 60-64. [1913. Demerara: printed by "The Argosy" Co., Ltd.]

Myiasis has been recognised for many years in British Guiana, though previous to 1902 it is not specially mentioned in annual reports, being included with many allied infections under the heading parasites. Since that date statistics from the principal hospitals of that country show a number of cases, varying from 4 to 12 per annum, the death rate from all forms of the infection working out at about 12 per cent. Of 15 cases treated in the Georgetown public hospital the following details are given: six were 50 years of age or over, four 40 to 50, four 30 to 40, and one between 25 and 30. In all debility was present and all possessed a history of a previous diseased condition.

The infecting fly in some of the cases was, according to WISE, as follows: (1) *Chrysomya macellaria*, (2) *C. violacea*, (3) *Calliphora erythrocephala*, (4) *Sarcophaga carnaria*. The commonest was the first, in the proportion of six to one. When the larvae of this fly are placed in a bottle containing cotton wool, pupae appear in 24 to 48 hours, while the full sized flies are hatched in another eight days.

A series of clinical observations in some of the cases observed is given. Diagnosis is usually made by the odour alone, the patient having a smell of putrid meat which is unmistakeable. This together with a sanguineous discharge from the nose or the presence of an inflammatory condition is very suspicious and can be verified by the actual finding of the larvae.

As regards treatment, early removal of the larvae is essential. When they are in the nose warm boracic lotion, 1 in 24, or carbolic lotion, 1 in 60, are recommended. When the sinuses are also affected surgical measures become imperative.

[The paper refers to one form of myiasis only. No mention is made of the larvae of *Dermatobia cyaniventris*, locally called mosquito worms.]

G. C. L.

HALL (M. C.) & MUIR (J. T.). A Critical Study of a Case of Myiasis due to *Eristalis*. — *Arch. Internal Med.* 1913. Feb. 15. Vol. 12. No. 2. pp. 193-202.

A child, five years of age, had been ailing for about ten weeks and was being treated for indigestion and constipation. Emaciation and anaemia were pronounced while nervous movements, such as twitching of the eyelids, grinding the teeth and convulsive movements of the limbs, were also present. The breath had a very foul odour.

A diagnosis of worms having been made, a dose of a proprietary remedy was given and a "rat-tailed larva," of the family *Syrphidae* of the order *Diptera*, was passed in the stool. The authors rightly point out that one has to be cautious in accepting the presence of fly larvae in faeces, the ease with which these may get into the faeces after it is passed being well known. They think however that their case was an authentic one, especially so as after the expulsion of the larva the child's health improved and the nervous symptoms disappeared.

They give a summary of seven published cases of the same infection known to them. These are as follows:—ODHELIUS's case of *Helophilus pendulus*; WAGNER's case of *Eristalis arbustorum*; LEIDY's case of *Eristalis* sp (nasal); RILEY's case of *Eristalis dimidiatus* and case of *E. tenax*; SHATTOCK's case of *E. tenax* and McCAMPBELL and CORPER's case of *E. tenax*. To this list should be added the case recorded in this paper [? *E. tenax*], the four cases from man recorded in the files of the Bureau of Entomology, and the one case from man and one from the cow (vaginal) recorded in the files of the Bureau of Animal Industry.

G. C. L.

CANDIDO (G.). *Anchilostomoanemia associata a Miasi intestinale*. — *Ann. Med. Navale e Colon.* 1913. Apr. Vol. 1. No. 4. (Anno 19.) pp. 394-407. With 1 fig.

It was not possible to breed out the fly from the larvae found in the faeces as they were always dead. Their macroscopical and microscopical appearances, however, were sufficient to enable one to say that they were those of *Calliphora vomitaria*, one of the common blow flies or blue bottles. Purgatives and antiseptics were successful in the treatment. [Infections with this fly have fairly often been described. The eggs are supposed to be ingested with vegetables or other food.]

G. C. L.

JONES (Glenn I.). **Hepatic Abscess (Non-Amoebic) and Gastro-Intestinal Myiasis.**—*Jl. Amer. Med. Assoc.* 1913. Oct. 18. Vol. 61. No. 16. p. 1457.

The case was a fatal one of a single liver abscess, destroying the entire right lobe of the liver, but containing no amoebae. While the patient was being operated upon, gastric lavage was practised with a view of ameliorating the hiccough and anorexia which were present. The stomach contents contained between 20 and 30 living larvae of *Musca domestica*. Another case of intestinal myiasis was accidentally encountered at the Jolo Hospital, Philippine Islands, where the present case had been before his operation.

[The fact of the abscess being a single one and occurring in the Philippines is certainly suggestive of an amoebic origin. The coincidence of the larva of flies in the stomach is interesting, but means nothing.]

G. C. L.

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## TROPICAL DISEASES BUREAU.

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## MALARIA.

BAHR (P. H.). **Malaria in Kurunegala.**—8 pp. Fcap. With 11 photographs and 1 map. (Report dated April, 1913, forwarded from the Colonial Office).

Kurunegala is a town situated in one of the rice-producing districts of Ceylon, and this most interesting report, which is furnished with an excellent map and several good and instructive photographs, gives an account of what was found in the course of a seven days' survey of the town and vicinity in February and March of this year. Some of the information has also been derived from the more extended experience of the local native medical officer.

The weather at the time of Dr. Bahr's visit was abnormally dry, the paddy fields lay fallow and there were few mosquitoes about and little actual fever in evidence. A great deal of valuable information was, however, collected which may be considered under different headings and in some detail.

(a) *The Main Species of Mosquito conveying Malaria.*

In the following list, which gives all the various species of *Anopheles* found in Kurunegala in the order of their frequency, those known to be carriers of infection are printed in heavy type:—*Anopheles barbirostris*, *rossii*, *sinensis*, *culicifacies*, *fuliginosus*, *jamesii*, *punctulatus*, *maculatus* *vel* *Willmori*, *albirostris*, *listonii*. It is chiefly in India and Malaya, that the five [six?] "carrier" species were found to be so. It was not possible to obtain this proof at Kurunegala for there are special difficulties regarding the experimental transmission work in Ceylon. These are stated by the author whose assistant found adult anophelines during the daytime in houses and bungalows in the town. They were never discovered in station bungalows, where malaria is very prevalent, and this is thought to be due to the mosquitoes retiring to neighbouring jungle during the day. Bahr was unable to find any anophelines in the daytime in the houses of Habage village [see map].



(b) *The Areas in which these Anophelines breed.*

1. *Paddy fields*.—These extend over an area of about 300 acres within town limits. The majority are only operative at certain seasons when flooded and especially when the water is subsiding. Some, irrigated from town wells and from a tank, are nearly always under water.

2. *Tanks*.—A main and bathing tank. Both are stocked with fish and consequently free from larvae.

3. *Irrigation Channels*, when badly graded or dammed up by natives.

4. *Rock Pool*, due to rain water and heavily infested. Its condition improved when fish were put in it.

5. *Borrow Pits*, now, thanks to the anti-malarial operations, almost inoperative.

6. *Freshwater Wells*. 180 in number, 18 of which contained a few larvae. Larvivorous fish having been introduced they can now be disregarded.

7. *Rock Stream* with stagnant pools in the dry season. *A. barbirostris* found in them and one specimen of *A. maculatus vel willmori*. The natives dam the stream to form a bath for their bulls, a practice to be forbidden.

8. *Water Channels on each side of Railway Cutting*. These are not kept clean, and are overgrown with *Spirogyra*, etc., forming excellent feeding grounds for larvae. This and the continually flooded paddy fields are most to blame.

The following table is interesting.

Nature of Breeding Ground.			
	Paddy Fields.	Rock Pool.	Railway Cutting.
<i>A. barbirostris</i> ... ..	19	47	—
<i>A. rossii</i> ... ..	26	42	14
<i>A. sinensis</i> ... ..	15	7	—
<i>A. culicifacies</i> ... ..	3	—	7
<i>A. fuliginosus</i> ... ..	1	—	—
<i>A. jamesii</i> ... ..	2	—	—
<i>A. punctulata</i> ... ..	1	—	—
Total examined ...	67	96	21

Four species of larvivorous fish are found in the flooded paddy fields:—

- “(a) ‘Pathia’ (*Barbus stigma*), a most efficient larvivore;
- (b) ‘Dandie’ (*Rasbora daniconius*);
- (c) ‘Sudaya’ (*Dania malabaricus*); and one, a bottom feeder;
- (d) ‘Ahirawa’ (*Lepidociphalichthys thermalis*).”

They are carried to the fields from the tanks by irrigation channels. It is thought that some are carried to remote blocks as spawn on the feet of water birds. Provided there is sufficient water they seem to multiply. It appears they only feed on live larvae and many of the latter have therefore adopted the habit



**RAILWAY CUTTING.** Water course flowing on each side of the permanent way ; supplying engines with water. These channels are blocked with weeds, from amongst which larvae of *A. culicifacies* and *A. rossii* have been taken



**FLOODED PADDY FIELDS.** (Opposite the Railway Station.) Water supplied from Wenaruwewa tank. This picture shows in the foreground the pools formed by the hoof marks of cattle , in these *Anophelines* breed.



of shamming dead and may thus escape. Larvae however exist along with the fish, and Bahr's explanations of this fact are:—

"(a) Larvae are generally found in the 'seepage' water which has filtered through the bunds leaving the fish behind.

"(b) In the larger pools which form in the paddy fields the larvae are found at the edges of the pools, while the fish keep the centre.

"(c) Holes formed by the feet of cattle (*vide* photo), and into which the fish are not able to enter, are ideal breeding places, and in these anopheline larvae are invariably found."

Frogs abound but do not appear to feed on the larvae at all. There is a note to the effect that the commonest larvae with cannibalistic propensities are those of *Culex tigripes*, a common mosquito in the roofs of native houses.

### (c) *The Effect of these Areas on the Prevalence of Local Malaria.*

The degree of infection in children under 14 years of age was taken as an index of the general population and a spleen survey was undertaken and microscopical blood examination conducted. In the 435 children examined the total spleen rate was found to be 34.7 per cent. and was higher in males than in females. [The variations met with in different localities are best studied in the map to which notes have been added briefly explaining the conditions].

The blood of all the 435 children was systematically examined. Malarial parasites were only found 45 times. The mean parasite rate for the whole town was 10.5 per cent. At H [see map] where the spleen rate is highest the parasite rate is low, an anomaly accounted for by various factors. Save in two instances the infection was slight, so possibly cases were overlooked. None of the children had fever at the time of the examination and indeed they looked remarkably healthy. Gametocytes were the forms chiefly found. The quartan parasite predominated though all three species of malarial parasite were present. Crescents were only found on three occasions. Of special interest is the fact that parasites were found in the blood of 15 children in whom *no concomitant enlargement of the spleen could be detected*.

There are some notes on the results of systematic treatment with quinine, from which it would seem that it effected considerable improvement in the spleen rate. Bahr's recommendations may be quoted in full as they apply in large measure to many other malaria-stricken localities in the Tropics.

"(1) Abolition of paddy fields within town limits.

"(2) Systematic treatment of all school children with quinine during their attendance at school. This practice to be continued for two years after the abolition of paddy fields. Enforcement of the Town Schools Ordinance of 1908.

"(3) Cementing all drains and waterways in the town, and more especially the streams in the railway cutting. These drains, after being cemented, must be systematically cleansed of all vegetable growth.

"(4) Stringent rules to prevent natives from blocking up streams and irrigation channels for the purposes of washing cattle or of catching fish.

"(5) Bund of channel from Wenoriwewa tank which supplies engine sump and paddy fields near station to be kept in repair to prevent leakage.

"(6) All irrigation channels should be properly graded so as to serve as outlets for storm water. In this respect the irrigation channel behind the station yard requires attention.

"(7) No borrow pits to be excavated within town limits without permission of the Local Board, who alone ought to be responsible for this work.

"(8) Rules to enforce adequate disposal of recently opened and discarded coconuts for drinking purposes. Owing to the abundance of firewood in Kurunegala, these shells are not used for fuel as elsewhere. In the spacious hollow water collects and forms an ideal breeding ground for all species of mosquito.

"(9) Enforcement of a 'Cattle Straying Ordinance.' Could cattle be prevented from straying into paddy fields, the 'goiyas' would then be able to reside at a greater distance from their work. At present, although realizing in some instances that these situations are unhealthy, they are unable to move further away on account of the damage to their crops arising from stray cattle."

It is proposed to substitute the planting of coconuts, a lucrative occupation, for the rice cultivation. Some useful appendices complete the report.

[This paper is well-nigh a model one of its kind but it is unfortunate that the total population is not stated, and that nothing is said about the influence of mosquito migration or, save as regards a brief note on rainfall, concerning the meteorological conditions. The report, however, is admittedly only of a preliminary nature. It cannot fail to be of great service to those whose duty it is to cope with malaria in irrigated districts].\*

A. Balfour.

JAMES (S. P.) & GUNASEKARA (S. T.). **Report on Malaria at the Port of Talaimannar.**—11 pp. F'cap. Illustrated. 1913. Colombo: printed by H. C. Cottle, Government Printer. [Ceylon. xxxiv. 1913.]

Talaimannar is the terminus in Ceylon of the new railway to India. It is on the small island of Mannar, a map of which appears in the Report. The district has always been regarded as one of the most malarious in Ceylon and is liable to flooding, the subsoil water being very near the surface. The yearly rainfall averages about 37 inches, most of the rain falling from

\* The author has furnished further useful particulars which may be tabulated as follows:—

Size of area under consideration=about 4 square miles

Area of rice fields within town limits=500 acres.

Area of inhabited part of town (bazaar)=15 acres.

Area of large irrigation tank=104 acres.

Population (1911 census)=8,163

Average temperature=79·8° F.

Mean Annual Rainfall=80·52".

Rainy months=April, May, October and November.

Height of rocks behind the town=Over 800 feet. These somewhat influence the temperature.

Kurunegala (pronounced "Corrygalle") is the capital of the North Western Province of Ceylon. At present it is a railway centre and is also the centre of an important agricultural district (rice and coconut cultivation). It will become more important as a labour-distributing centre when the Mannar Railway is completed. The maximum malarial incidence generally occurs three months after the heaviest rainfall and about two months after the hottest period of the year. In 1911 a total of 100 deaths was attributed to malaria in the district. A more elaborate report will be forthcoming at a later date.

A. B.





October to January. The mean annual shade temperature is nearly 83° F., but variations occur both in rainfall and temperature, and the authors state that in the tropics it is usually the case that abnormal meteorological conditions of any kind are adverse to the population. In the Mannar district the latter are for the most part wretchedly poor and ill-fed Ceylon Tamils and Moors engaged in agriculture and fishing. Statistics show a rapidly falling birth-rate, a high death-rate and a heavy infantile mortality both in the Mannar district and in the Mannar island division. The chief cause of sickness and mortality is malaria, and when speaking of respiratory diseases the authors mention that in India there is a close association between malarial infection and fatal lobar pneumonia. A considerable proportion of the deaths in Mannar being ascribed to malarial cachexia, the authors thought it well to exclude kala-azar. This they were able to do. A spleen-rate and a parasite-rate were worked out. The latter was found to be considerably lower than the former (36 per cent. as against 58 per cent.). Of the parasites found 71 per cent. belonged to the quartan type. It seems clear that malaria is accountable for the deplorable state of the district.

Other interesting particulars are furnished. As regards the anopheles factor, *Myzomyia culicifacies* is probably the mosquito chiefly to blame but seven species of anopheles occur. The important part played by the social and economic conditions of the community is insisted upon. Amongst other things these may favour relapses so that a very few anopheles may suffice to maintain a high parasite infestation. The question of the introduction of non-immune immigrants is fully discussed as is the influence of the rainfall. A recurring epidemic is usually superimposed annually on the endemic malaria and the definite relationship of this epidemic prevalence to the rainfall is shown graphically by means of a chart. Before passing to the question of preventive and curative measures the authors uphold the view that unless attempts are made to prevent or minimize the effects of the floods other methods are unlikely to prove successful. The chapter on prevention is both interesting and instructive and will be found specially valuable by those engaged in railway extension work in the moist tropics. It is, unfortunately, impossible to review this section in detail here. A matter, however, meriting more than a passing reference is the question of housing for a European Staff. According to the authors the following principles should guide those responsible.

"(1) The houses should be, as a rule, square, and may be of one or two storeys.

"(2) They should be raised from the ground, preferably on an extensive plinth of concrete surfaced with cement.

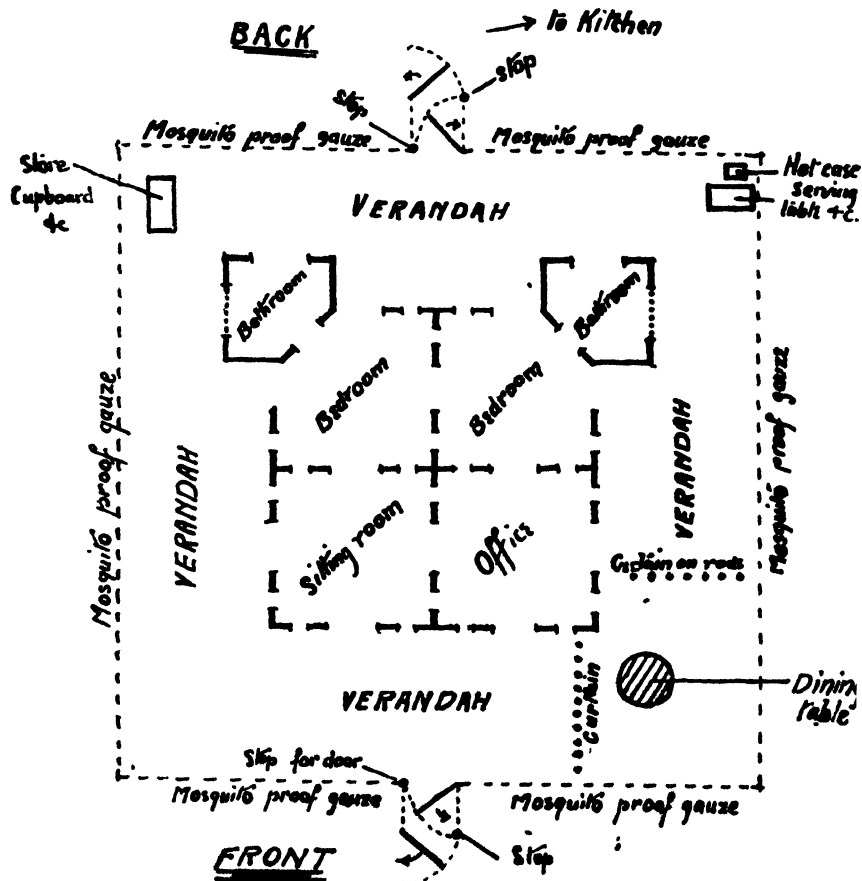
"(3) The whole area of the plinth should be utilized for the erection of a large mosquito-proof 'shed,' inside which the house proper will be built.

"(4) The 'shed' consists of the following parts:—(a) Pillars of iron, stone, or wood, erected at the corners and along the outer limits of the plinth to support the roof; (b) between the pillars a dwarf wall, and upon it a framework filled in with mosquito-proof copper wire gauze of 18 meshes to the inch; (c) at two places in the skeleton walls double doors, mosquito-proofed, and both opening outwards. They should be made to close automatically (N.B.—It is essential to the success of mosquito-proofed houses that all doors should open outwards); (d) a ceiling of wood



or of asbestos material, resting on and closely applied to the top framework and girders in such a manner that the roof is completely shut off from the remainder of the shed; (e) a sun-proof and rain-proof roof, which is ventilated, and of which the eaves project as far as possible beyond the framework and are not provided with gutters—the rain dripping from them being allowed to fall into a concrete drain laid all round the house; (f) the result is a large square mosquito-proof structure with walls of mosquito-proof wire gauze, a ceiling of wood, and a floor of cement, and the whole covered by a roof; (g) all that now remains to be done is to reserve a broad space all round inside the structure to serve as a verandah, and then to partition off the remaining space into rooms. It is not necessary for these partitions to be very solid structures, and they should be opened wherever possible by large arches and windows. Doors to the arches are not necessary. It is important that no partition should be built which will cut off a through draft of air from front to back and from side to side of the house. (h) The house should be orientated so as to obtain the greatest advantage from the winds of the south-west and north-east monsoons."

The accompanying plan is reproduced as it is likely to be useful. There is a careful criticism of the faulty type of house



Plan of four-roomed house suitable for residents in malarious districts.

(The large openings are archways, the small ones are windows.)

previously adopted. The reason why doors should not be made to open inwards into rooms is because mosquitoes resting on the

outside of such doors will be brushed into the room along with the person entering—a point worth remembering but often forgotten. A mosquito catcher is recommended as a profitable asset.

A final note states that the borrow pits are not so much to blame as has been supposed, but, quite apart from malaria, they are very insanitary and should be abolished. As the port is likely to become important “hydraulic fill” operations may eventually be required on the lines of those employed at Panama and elsewhere.

A. B.

**SARKAR (Satosi Lal).** *Investigations into the Incidence of Malaria in the Town of Arambagh, Hooghly District.*—*Indian Med. Gaz.* 1913. Sept. Vol. 48. No. 9. pp. 342-346.

This paper embodies the results of an enquiry at Arambagh into the relative liability of Hindus and Mohammedans as well as of different Hindu castes to malarial infection. This was carried out by conducting a spleen census of children between two and twelve years of age. In the different wards of the Municipality 1,721 male and female children were examined while for comparison of race and caste 379 Mohammedan and 1,338 Hindu children came under observation. The latter part of the investigation was important because it is generally believed that, in Bengal, the Hindu populations are being wiped out by epidemic malaria at a greater rate than the Mohammedan populations. The author found scarcely any difference between the spleen rates for Hindus and those of Mohammedans. [Unfortunately he does not classify the degree of splenic enlargement recognised nor explain fully how the enquiry was conducted although it would seem that kala-azar cases were excluded]. He thinks that the table comparing race and castes shows (1) that the prevalence of malaria in a caste is in accordance with the economic conditions of the caste. The better these are the greater the resistance to infection; (2) that the castes which perform out-door work are less liable than those whose occupation lies indoors.

A. B.

**SATYARANJAN SEN.** *Some Observations on the Aetiology of the Malaria in Bengal.*—*Indian Med. Gaz.* 1913. Aug. Vol. 48. No. 8. pp. 303-305.

The author seeks to show that in Bengal the conditions commonly considered as favouring the prevalence of malaria, *i.e.*, the presence of jungles, water-logging of the soil and defective drainage, the existence of partially dried up rivers and canals and of pits, holes, tanks and borrow-pits, apparently do not influence the distribution of the disease. He bases his argument on “spleen censuses” in children under twelve carried out in different localities. He found villages near each other in which malaria varied in degree and intensity independently of such causes. There is less malaria in the new villages where there are better houses and a higher standard of comfort.

A. B.

ADIE (J. R.) & Mrs. ADIE. **Note of an Inquiry into Malaria and Mosquitoes in the Kashmir Valley.**—*Indian Med. Gaz.* 1913. Sept. Vol. 48. No. 9. pp. 341-342. With 1 map.

This paper is practically the same as that reviewed in this *Bulletin*, Vol. 2, p. 315, but a map showing the mountains and rivers connected with Kashmir is appended.

A. B.

BRÜNN (W.) & GOLDBERG (L.). **Die Malaria Jerusalems und ihre Bekämpfung.** [Malaria in Jerusalem and its Control.]—*Zeitschr. f. Hyg. u. Infektionskr.* 1913. Aug. 27. Vol. 75. No. 2. pp. 209-235. With 25 text-figs.

Jerusalem is a city of, approximately, 70,000 inhabitants, comprising about 45,000 Jews, 10,000 Mohammedans and 15,000 Christians. There are also many pilgrims. Although 750 metres above sea level and possessed of a good climate it is a most unhealthy spot. This is due to the great poverty of the majority of its populace and to the slight interest in hygienic matters hitherto shown by the Turkish authorities. The authors give an account of the truly deplorable sanitary, or rather insanitary, state of the city, and especially of the old town. It is illustrated by numerous and excellent photographs. Thanks to the philanthropy of Nathan STRAUS of New York it has been possible to institute an anti-malarial campaign in Jerusalem and the authors give a most interesting account of its prosecution.

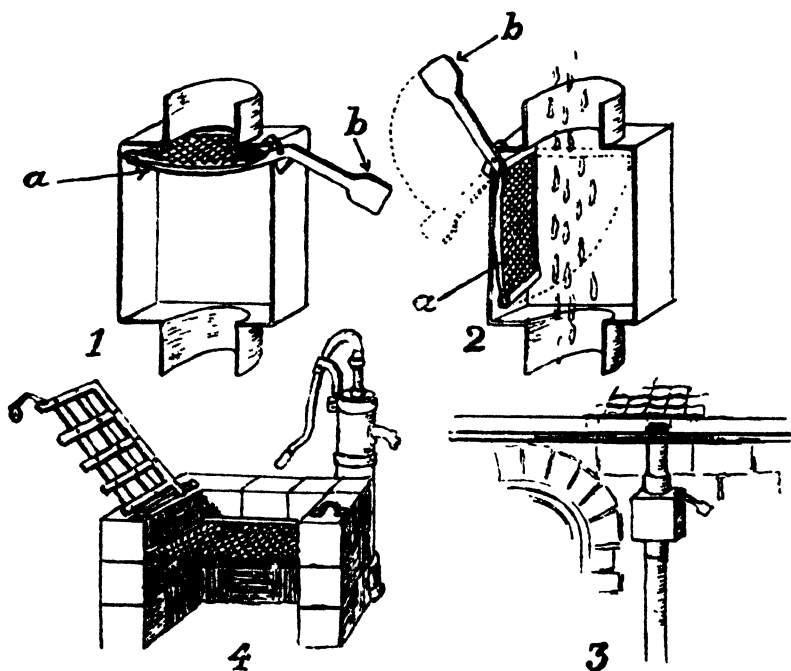
Naturally it is chiefly of local interest, but much that they say applies to other towns in the Near East.

In the summer *Anopheles* were found breeding only in the water cisterns, which are such a feature of the city and which are most carefully described and figured. Although there are several large open water basins *anopheles* larvae were never found in them. In two instances their absence was attributed to the presence of fish. Only in the pool of Bethesda were larvae (*Culex*) found. In the winter and spring, however, pools and puddles form after heavy rain and these harbour numerous mosquito larvae, including *anopheles*. The cisterns are of a peculiar type and serve for the storage of the rain water which at the present time constitutes the drinking water supply for the inhabitants. These cisterns are sometimes very large, 4, 5 or 6 metres deep, are built of stone and are situated in the courtyards or the basements of the houses. They are frequently in a very bad state of repair, and are rarely provided with pumps, the water being usually obtained through a well mouth by means of a bucket. The well mouth is generally enclosed in a raised stone coping and is very often only protected by an iron grating through which mosquitos readily gain access to the water. In some instances solid hinged well-fitting metal lids are provided, but both lids and grating are in use more to prevent the precious water being stolen than from any sanitary consideration. Owing to various defects all manner of filth frequently finds its way into these cisterns. In addition to the mosquito work the authors instituted a ~~spoon~~ <sup>spoon</sup> survey from which they conclude that out of every five of the inhabitants three show splenomegaly. Full details are

given regarding 616 cases examined. Of these 365 or 59 per cent. showed splenic enlargement, and these are classified according to the grade of splenomegaly present, four degrees of enlargement being recognised. From August 20th to January 15th an examination of the blood for malarial parasites was made in the case of 2,055 persons. In 442 or 21·5 per cent. the result was positive. In other words every fifth inhabitant during summer and autumn harbours malarial parasites in his blood. The following table gives the types of parasite found.

70 cases	=	16 per cent.	Benign tertian.
94 „	=	21 „ „	Quartan.
265 „	=	60 „ „	Tropical (malignant).
13 „	=	3 „ „	Mixed infection.

The cases are also classified according to age and locality. The authors point out that the only way of combating malaria on a large scale in Jerusalem is by a crusade against mosquito larvae, although in certain parts destruction of the imagines by fumigation or spraying may be practised with advantage. Quinine prophylaxis for the whole town is out of the question, being too costly and too difficult to carry into effect properly. At the same time, where possible, it is not to be neglected. The mosquito campaign practically resolves itself into a proper covering of the rain-water



Figs. 1, 2 and 3.—Apparatus for fixing in rain-pipes to prevent ingress of mosquitoes to cisterns.

Figs. 1 and 2 are sectional views of the apparatus illustrating mode of working. (In Fig. 2 the diagram is transposed to shew the valve open.)

*a*, valve of zinc-coated wire netting; *b*, weighted arm to keep *a* in horizontal position, thus closing the opening. The weight of the rain on *a* causes the valve to open; when the rain ceases the valve is automatically closed again by the counterpoise *b*.

Fig. 3 shews apparatus in position on rain-pipe.

Fig. 4.—Method of screening large air vents or well mouths.

cisterns, petrolage for those containing dirty water, and also for all pools and puddles. Each rain water cistern should be provided with a pump, the well mouth being tightly covered and the rain pipe carried through the cistern wall and properly built into it. All holes must be closed up. The authors have invented and figure an apparatus which can be fixed in the rain pipes to prevent the ingress of mosquitoes to the cisterns while presenting no barrier to the rain. They have also devised a method of screening the large air vents or well mouths which serve some of the cisterns.

As they point out, these measures should greatly improve the general health of the inhabitants. In addition to malaria, dengue, enteric fever and dysentery should all diminish. The ideal sanitary reform for Jerusalem would be the provision of a proper piped water supply. At present there is no hope in this direction so that the methods described must be adopted in their entirety.

A. B.

LEGER (Marcel). *Le Paludisme en Corse.*—*Ann. Inst. Pasteur.* 1913. Sept. Vol. 27. No. 9. pp. 765-793.

This is to all intents and purposes the same paper that was reviewed in this *Bulletin* Vol. 2, p. 215. It should, however, be noted that an examination for infected Anophelines was made in the region of Aleria which is the most malarious in Corsica. Of 295 *Anopheles maculipennis* collected from three different localities and examined by SERGENT's method only seven were found infected, a percentage of 2.37.

A. B.

FALCIONI (Domenico). *Sulla Localizzazione della Malaria alle Abitazioni.* [On the Restriction of Malaria to Habitations.]—*Policlinico.* Sez. pratica. 1913. Aug. 3. Vol. 20. No. 31. pp. 1119-1122.

The author points out that in all regions where malaria prevails its incidence in particular houses or streets of a town can be demonstrated, showing that infected mosquitoes never move far from spots where they can find human beings on whom to feed. Shepherds in remote parts of the Roman Campagna can sleep out at night with impunity in spite of the prevalence of mosquitoes, because there are no human beings affected with malaria in the vicinity, from whom the parasite could be conveyed.

A. B.

PEALL (P. A.). *An Inquiry into the Effects of Malaria upon the Health of our Mine Native Labourers.*—*Med. Jl. of S. Africa.* 1913. August. Vol. 9. No. 1. pp. 6-9.

As the native labour for the mines in Southern Rhodesia is wholly recruited from so-called "Tropical Boys," i.e. labourers coming from districts within the tropic belt, the author thought it advisable to conduct an enquiry into the incidence of malaria amongst these labourers and also into the effect of chronic

malarial infection upon their blood-forming organs and their powers of resistance to other diseases. Out of a total of 102 blood films examined parasites were observed in 46 cases=45 per cent. Many of the cases of malaria were undoubtedly relapses due to change in residence, fatigue and chill. Particular attention was paid to the presence and character of the degenerative changes in the red cells and more especially to the occurrence of the large, pale, semi-lunar red corpuscles first described by BRUMPT. Degenerative changes were found in 68 films=66 per cent. The relative proportion of large mononuclear leucocytes and the presence of malarial pigment in these cells were also noted. The author is of opinion that the blood degeneration was evidence of a chronic malarial infection and that the degenerative changes, so induced, very probably had a debilitating effect upon the general health and efficiency of the natives, rendering them less resistant to bacterial invasion. He found that an adequate administration of quinine caused a speedy disappearance of the half-moon red cells from the peripheral blood, and he advocates the exhibition of quinine prophylactically combined with tonic treatment in the case of all labourers during their sojourn in detention camps and while travelling to the mines.

A. B.

GOBERT (E.). *Quelques Aspects du Problème Antipaludique en Tunisie* (1912).—*Arch. Inst. Pasteur Tunis*. 1913. No. 1 2. pp. 121-128.

This is a paper by the chief of the Anti-malarial Service in Tunis giving an account of the condition of several districts which are heavily infected by malaria although some of them differ greatly in their general features. The article possesses only local interest save perhaps the part which deals with the construction of the large drainage canal in La Mabtouha, an undertaking which has proved of great benefit both from an agricultural and sanitary standpoint. A reference is given to a memoir on the subject written by the engineer in charge of the work. It is evident that the campaign against malaria is being actively prosecuted in Tunis, all known methods being employed though, in some districts, quinine prophylaxis is the only feasible measure.

A. B.

O'CONNELL (Matthew D.). *The Meteorology of Malaria*.—*Jl. Trop. Med. & Hyg.* 1913. Sept. 1. Vol. 16. No. 17. pp. 257-260.

The author suggests that certain meteorological conditions by themselves, in Calcutta and places with a similar or nearly similar climate, can, at certain seasons of the year, produce a fever resembling malaria. Further he apparently considers that such conditions may be responsible for the intermittent pyrexia, haemolysis and splenomegaly which are so characteristic of malaria and which are usually attributed to an unknown toxin. [This, however, as BROWN has indicated may be hematin—see this

*Bulletin*, Vol. 2, pp. 341, 342.] In support of his view O'Connell has compiled tables showing hourly atmospheric conditions at Calcutta in March and September, 1912, and they are compared with atmospheric conditions which raised body temperature in Lancashire cotton-weaving sheds. [As, however, no one is likely to accept the author's views in their entirety it is perhaps unnecessary to review the paper in detail.]

A. B.

KNAB (Frederick). *Anopheles and Malaria*.—*Amer. Jl. Trop. Diseases & Preventive Med.* 1913. Sept. Vol. 1. No. 3. p. 227.

The author corrects some errors in the paper which was reviewed in this *Bulletin* Vol. 2, p. 317. The corrections do not apply to any of the points considered in that review.

A. B.

## DIAGNOSIS.

FRÓES (João A. G.). *The Rapid Diagnosis of Malaria*.—*Jl. Trop. Med. & Hyg.* 1913. Sept. 1. Vol. 16. No. 17. p. 272.

The author gives details of a modified CROPPER's method (*British Medical Journal*, 1912, Apr. 20, p. 890) of staining thick blood films in malaria. His technique is as follows:—

(a) Make very thick films with five large drops of blood.

(b) When the blood has dried, stain it for two minutes with Gasis's solution:—

Methylene blue	...	...	...	1 grm.
Hydrochloric acid	...	...	...	0.5 cc.
Alcohol	...	...	...	10 „
Distilled water	...	...	...	90 „

(c) Wash carefully, dry and examine with  $\frac{1}{12}$ " oil immersion. The good results obtained are said to be due to the fact that the staining takes place at the same time as the acid solution acts on the red cells dissolving their haemoglobin. The whole malarial parasite is visible, not only the pigment as in the case of CROPPER's method.

A. B.

ACTON (Hugh W.) & KNOWLES (R.). *The Diagnosis of Latent Malaria*.—*Indian Jl. of Med. Research.* 1913. July. Vol. 1. No. 1. pp. 167-176. With 9 charts.

This is the paper reviewed in the *Bulletin* Vol. 2, p. 321. The charts, nine in number, have now been added and will be found useful by anyone studying the article.

A. B.

## CLINICAL AND TREATMENT.

BATES (J. P.). *A Review of a Clinical Study of Malarial Fever in Panama*.—*Jl. Trop. Med. & Hyg.* 1913. Aug. 15. Vol. 16. No. 16. pp. 241-245.

This is the fourth section of the above review and is concerned with the question of relapse. The author points out the difficulties attending a research upon relapse in malaria. It is not

easy to exclude reinfection as shown, for example, in the case of three persons who suffered from apparent relapses until their quarters, which had been recently screened, were thoroughly fumigated when they at once ceased to get fever. At the same time it was possible to obtain a certain number of cases suitable for prolonged observation. Three chief types of relapses are mentioned:—

1. Those occurring in children, always impatient of control, and in adults whose duties force them to commence work too quickly after subsidence of the fever. Here the relapses occur at short intervals and would seem to bear some relation to the thoroughness of the quinine treatment in the primary invasion or in the succeeding attacks. If the treatment is efficient relapse does not occur.

2. Those in people living in malarious countries who may suffer from several attacks of malaria and who finally reach a stage where they can live in a malarious country in apparent good health but relapse as soon as they go to a cooler latitude or cooler climate even when they have been taking quinine prophylactically. Such relapses may be separated from each other by periods of five, six, or eight months and sometimes even longer.

3. A doubtful group where the relapses are separated by periods of several years. Bates does not believe that these are true relapse cases but admits that the question is still *sub judice*.

He goes on to consider the four theories hitherto advanced in order to explain the mechanism of relapses, *i.e.* (a) residual parasites which have survived the effect of quinine and continue to develop in the usual asexual cycle (ROSS); (b) parthenogenesis of sexual forms (SCHAUDINN); (c) intra-corpuseular conjugation (CRAIG); (d) sexual development in the human host *e.g.* fertilisation of female crescents by the male crescents and subsequent segmentation (THAYER). The last is based on the work of ROWLEY-LAWSON.\* The author discusses all these views at some length and in an interesting manner, citing especially the work of DARLING at Panama in 1909 on malaria "carriers." The original must be consulted by those interested, but with the evidence before him and as a result of his own observations Bates agrees with ROSS that every circumstance favours the view that residual parasites are responsible for relapse. In the case of the other theories, even if the phenomena described do occur and have been correctly interpreted, their relation to relapse is merely conjectural. On the other hand it is known that the parasite of the asexual cycle can survive over indefinite periods of time, and it seems reasonable to suppose that, though they may be so few in numbers as not to be detected by our existing methods of examination, they can remain and multiply slowly until favourable conditions bring about their rapid increase and hence a true malarial relapse.

A. B.

\* *Jl. Experimental Medicine*. 1911. Feb. 1. Vol. 13 No. 2. p. 263.



SWELLENGREBEL (N. H.). **Schizogonie der weiblichen Gametocyten von *Laverania malariae* (Tropica-Parasit).** [Schizogony of the Female Gametocytes of *Laverania malariae*.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Aug. 4. Vol. 70. No. 3/4. pp. 179-181. With 1 coloured plate.

The author points out that Ross denies the occurrence of parthenogenesis in the female gametocytes of *Pl. vivax*, as originally described by SCHAUDINN in 1903. Swellengrebel asserts that while parthenogenesis of female gametocytes has certainly been found in the case of benign tertian infections it has never been described in the case of the quartan parasite, while, so far as the tropical parasite is concerned, the only positive observation is that of NEEB in 1909. [He seems to have overlooked papers by HARRISON and KARREWIJ in this connection.] Ross did not confirm NEEB's findings and, at the time, Swellengrebel agreed with Ross, but he has recently altered his opinion. This is the outcome of observations made in the case of a Chinaman admitted to hospital for malignant malaria at Deli on the east coast of Sumatra.

In this patient's blood, stained with Giemsa in such a way as to prevent overstaining of the chromatin, (one drop of stain solution in 1 cc. aq. dist. and allowed to act for 10 minutes) he found small rings, fully developed vegetative parasites, and all the stages up to normal schizogony, adult male and female crescents, and young crescents still enclosed in the red cells. These, together with the so-called schizogony of the female crescent, are illustrated by a coloured plate. He describes the ordinary vegetative [asexual] forms and their schizogony and then points out that the young crescents in the red cells can be distinguished from the asexual parasite by the fact that their cytoplasm takes on a less deep shade of blue and has a violet tinge, that their pigment is diffuse, and that their nuclei are less sharply defined. The plate illustrates the development of the young, spherical macrogamete first into endoglobular and then into free crescents. [It cannot be said, however, that the drawings are altogether convincing. Certainly that of a fully developed free crescent differs from one's usual conception of such a form even though it is true that the gametes of *L. malariae* do not always present a typical crescentic shape.]

There follows an account of the changes preceding schizogony in these young endoglobular forms. The chromatin becomes more or less scattered or arranged in a long streak. The streak or band divides to form two nuclear masses while the diffusely scattered chromatin breaks up into several portions. These eventually coalesce to some extent so that the mature crescent is multi-nucleated. The cytoplasm retains the violet-blue colour and the pigment remains diffuse. As a rule the crescents continue in the red cells but they may become free. Some of the chromatin usually remains in the form of a streak or of fine threads. Finally a stage prior to schizogony and like that found

in the ordinary asexual cycle is reached, the chromatin and pigment concentrating at the centre of the parasite but the concentration of pigment is never so marked as in the usual asexual cycle. This fact, the peculiar colour of the cytoplasm, the size of the parasites in relation to their host cells, and the peculiar arrangement of the chromatin differentiate this crescent schizogony from that of the vegetative forms. The author, like MAURER, has twice seen a multiple division of the chromatin in a *fully developed* crescent, but he cannot look upon this as evidence of schizogony for no further stage could be found.

He concludes by mentioning some of the possible fallacies already alluded to by NEEB. He found no evidence of a mixed infection and so can exclude a schizogony of the sexual forms of benign tertian or quartan parasites. The features described seem to put out of question an ordinary, vegetative schizogony while the number of nuclei found does away with the possibility of the appearances noted being due to a premature formation of male gametes, a phenomenon which has now and again been observed in the peripheral blood. Hence he believes he is justified in concluding that he has witnessed a true schizogony of young crescents in a case of infection by *L. malariae*.

A. B.

DA MATTA (Alfredo A.). **Pneumo-paludismo Asthmiforme.** [Malarial Pneumonia of an Asthmatic Type.]—*Rev. Med. de S. Paulo.* 1912. Oct. 31. Vol. 15. No. 20. p. 397.

The author was summoned to visit a youth of 18 years of age, who was suffering from a violent attack of fever, the temperature being nearly 40° C. In addition the patient was cyanosed, and the respiration asthmatical. Pain was complained of over the area of the right lung, and physical examination revealed dulness on percussion along with tubular respiration and other signs of congestion. The liver was also tender and enlarged, and the spleen palpably hypertrophied, while the urine contained albumen and an excess of urea.

As the locality in which the patient resided was malarious and no further information as to the nature of the case was forthcoming, a hypodermic injection of quinine was tentatively given, along with dry cupping to the region of the lung. On the next day, to the surprise of the reporter, the condition was found much ameliorated and the patient had a normal temperature. The blood was then examined and found to contain *Plasmodium falciparum*. The patient, being now in a condition to explain, related that he had had a similar seizure about two months previously, for which he had been treated by a medical man with various remedies without success. Seeing that the paroxysms showed a decided tendency to periodicity, the patient was persuaded by a friend to take certain capsules at the rate of three a day, whereupon improvement ensued. On the strength of this history the treatment with quinine was continued and the patient speedily recovered.

A. B.

DA MATTA (Alfredo A.). *Anosmia palustre*. [Malarial Anosmia.]  
—*Rev. Med. de S. Paulo*. 1912. Dec. 31. Vol. 15.  
No. 24. p. 483.

A married woman, aged 25, residing in the suburbs of Manaus, sought advice for pains in the head, violent at times and extending to the eyes and nose. They were always most intense on rising in the morning and lasted till 10 or 11 o'clock, after which the patient considered herself free. During the attack there was no sensation of fever, but great languor and indisposition to exertion. What most impressed the patient and led her to seek advice was a loss of smell, which manifested itself in the course of cooking meals. Physical examination of the nose and pharynx showed nothing abnormal, but the loss of olfactory power was undoubted. The temperature was normal and the blood contained no parasites. Nevertheless, the patient having suffered from malarial fever in the past, a diagnosis was made of malarial neuralgia of the fifth nerve with anosmia as a complication, and upon this hypothesis treatment was initiated with a dose of 30 centigrammes of hydrochlorate of quinine three times daily. The result was a brilliant success, both neuralgia and anosmia disappearing on the fourth day.

A. B.

AUDIAU. *Paludisme à Forme Hépatique*.—*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 454-455.

A soldier at Tuyen-Quang had quartan malaria which was apparently the sole cause of an enlarged, painful and congested liver. There was no previous history of dysentery. Quinine cured both the fever and the hepatic condition. Quartan fever is rare in French Indo-China and at first the diagnosis was difficult, as there was nothing to suggest the real cause and bacteriological examination proved negative.

A. B.

DAGORN. *Cas d'Hémoptyisie Paludéenne*.—*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 451-452.

A soldier at Hagiang, French Indo-China, with no evidence of tuberculosis or any pulmonary disease was found to be suffering from severe haemoptysis during attacks of tropical malaria. The haemorrhages were unassociated with cough and there was no sputum.

A. B.

LUGO-VINA Y CARTA (Nicasio). *La Tifomalaria y su Terapéutica Clínica*.—*Semana Médica*. 1913. Sept. 25. Vol. 20. No. 39. [No. 1028.] pp. 714-717.

\*The author recognises that a malarial infection may be superimposed on one due to the Eberth-Gaffky bacillus and *vice versa*. He deals with the difficulties of diagnosing such a hybrid condition. While he supports COLLIER's dictum to the effect that

microscopic blood examination and the Widal test can alone make the diagnosis certain, he thinks that in many cases "the clinical eye may be substituted for the microscopic lens." Many cases of typho-malaria would seem to commence with intermittent pyrexia and end in a similar manner. The termination in cases of recovery is most frequently by lysis. The author enters very fully into the question of treatment. He begins by exhibiting calomel alone or in combination with rhubarb and magnesia. Jalap may be substituted for the rhubarb. In the case of children he employs only calomel and magnesia. Later, for three or four consecutive days he administers quinine by intramuscular injection. Then follows a day without injection when a mild purgative water, such as Rubinat, is given. This is followed by three or four more quinine injections. When the temperature rises above 38° C. he gives antipyretics, more especially antipyrin together with bromide of sodium or potassium. He thinks intestinal antiseptics of value in certain cases and is accustomed to give benzonaphthol, or, if the urinary system requires attention in this direction, salol and benzonaphthol. These may be taken with bicarbonate of soda or along with a medicated water suited to the prevailing conditions. Thus if gastric symptoms predominate *Vichy* is indicated, if hepatic *Celestins*, if renal *Grande-Grille*. As the fever declines the author employs a mixture of arsenic and quinine and, where necessary, tonics and stimulants. One may note his use of iodine in potassium iodide locally to prevent septic infection after quinine injection, and, in lieu of baths, for sustained high temperature the employment of the wet sheet which has been soaked in aromatic vinegar. Children, after debilitating sweats, benefit from a shampoo with alcohol, or water and alcohol combined.

The use of tepid or cold enemata for reducing high temperature is also mentioned as is the question of diet and beverages. The value of sunlight and an open air treatment during convalescence receive due notice.

A. B.

ANNALES D'HYGIÈNE ET DE MÉDECINE COLONIALES. 1913. July-Aug.-Sept. Vol. 16. No. 3. pp. 781-783. [Clinique d'Outre-Mer.] **Extrait du Rapport du Médecin Chef de l'Ambulance de Langson.** [Malarial cachexia: pp. 782-783.]

An account of a spontaneous sub-capsular rupture of the spleen in a case of malarial cachexia. The patient, an European, had been seventeen years in Cochin-China, the last three of which had been spent in Tonkin. During this latter period he had suffered from severe headaches, loss of memory, broken sleep and some vertigo. These symptoms latterly increased in severity, he became troubled by nightmares and passed into a slight and intermittent state of low delirium. The clinical signs are mentioned, but there is nothing to note save that on December 8th there was a very marked rise of temperature and on the following day a violent dyspnoea ensued, the respirations being 60 to the minute. The heart failed; inhalations of trinitrine gave no relief and, though morphia eased the dyspnoea a little, the patient died an hour and a half after the beginning of the dyspnoeic attack.

The spleen, which presented an extensive, sub-capsular rent, weighed 590 grammes and its pulp was of the consistence of pap. The liver was much enlarged and the condition was plainly one of malarial cachexia.

A. B.

DAVIDSON (Wilson T.). **A Case of Spontaneous Rupture of the Malarial Spleen: Splenectomy.**—*Texas State Jl. of Med.* 1913. Sept. Vol. 9. No. 5. pp. 151-152. With 1 text-fig.

The causes favouring rupture of the spleen in chronic malaria are, in addition to hypertrophy, changes both in the parenchyma and the capsule. The pulp becomes soft and diffuent, the capsule distinctly sclerotic. Adhesions to neighbouring organs also tend to form. The condition is rare, only three cases out of 30,000 cases of malaria being reported from the Colon Hospital during a period of eight years. The author describes a case from the Philippines under his care in Texas where splenic rupture took place without any definite trauma having occurred. Splenectomy was performed and the patient recovered. It is pointed out that in addition to the symptoms of internal hæmorrhage one is led to a correct diagnosis by the presence of pain in the splenic region and of flatness upon percussion in the more dependent portion of the abdominal cavity. Stress is laid on the importance of the symptom noted by BALLANCE, *i.e.*, both flanks are dull upon percussion when the patient lies on his back. When he turns on his *left* side the right flank becomes resonant because the blood is free. When, however, he turns on his right side the left flank does not become resonant owing to the blood clots held by the intestinal folds. When this symptom is present a positive diagnosis of splenic rupture can be made. If it is absent, there may still be an injury of the spleen. A list of symptoms following splenectomy, but gradually disappearing, is given. Of these may be mentioned a tenderness along the long bones, said to be due to active changes taking place in the marrow which is assuming the function of the spleen.

A. B.

BATES (John Pelham). **A Review of a Clinical Study of Malarial Fever in Panama.**—*Jl. Trop. Med. & Hyg.* 1913. Oct. 1. Vol. 16. No. 19. pp. 297-301.

Continuing his dissertation on malaria the author has now reached the subject of treatment. He points out that in estimating the effect of quinine one must take into consideration the tendency of cases of malaria to recover naturally. Two cases, apparently the same in every respect, may run very different courses, the one recovering without treatment, the other tending to show pernicious symptoms. In both temperate and tropical climates most cases tend to terminate favourably in from 10 to 15 days or at the most in 20 days. Thirty grains would seem to be the average daily dose of quinine generally recommended but the drug must be increased in quantity to meet the gravity of the infection. Bates finds that it is perfectly safe to give much

larger doses than are usually advised, and in grave cases in Panama the doses given largely exceed the 40-45 grains mentioned as a maximum in the text-books. The routine treatment there adopted is to give 20 grains in solution on admission and thereafter 30 grains daily. For the therapeutic test 45 grains a day are sometimes given for one or two days and then the usual 30 grains daily. In grave and pernicious cases it may be necessary to increase the dose to from 60 to 90 grains. Indeed Bates has given as much as 120 grains in the first 24 hours, but it is important to note that *massive doses must not be continued longer than 24 hours*. As a matter of fact cases requiring more than 80 grains within the first 24 hours are usually fatal within this period. If massive doses are given the quinine should be reduced after 24 hours to 45 grains. This dose may be continued for 24 hours or 48 hours and then a further reduction to the usual 30 grains is indicated. In cases where the peripheral blood is freed from parasites, and this means that the circulation generally has been cleared of them, but where the gravity of the symptoms continues to increase, the quinine should be rapidly reduced in quantity as in such cases it does no good and will not prevent death. The author gives the following as indications for increasing the dose of quinine:—

1. A large number of parasites in the peripheral blood. It must, however, be remembered that the mere number of parasites present is no guide to the severity of the case.

2. The characteristics of the parasite present. For example, in quotidian and subtertian infections the occurrence of presegmenting and segmenting forms in the peripheral blood is usually a grave symptom.

3. The state of the blood. When the blood exudes with great difficulty from the pricked ear or finger and when it is dark in colour, clots quickly and spreads unevenly over the slide one suspects blocking of capillaries or malarial toxæmia. In such cases intravenous injections of 20 grains in 10 cc. saline solution are indicated. The author has no experience of quinine given intravenously in large dilution as employed by JAMES.

4. What may be called "pernicious aura" [A.B.], *i.e.*, mental aberration, inability for consecutive thought, defective memory as regards the details of the illness, sometimes moroseness or mental dulness, vague wanderings away from the bed, &c.

When these signs appear push the treatment even if there is only a moderate infection in the peripheral blood. Give 45-60 grains in the first 24 or 48 hours.

The time of day for the administration of quinine is considered. The author is partial to a dose twice daily, both doses being given in the forenoon, say 15 grains at six and another 15 grains at eleven o'clock. Such a method is less troublesome for the patient and ensures the quinine being in greatest concentration in the blood about the time sporulation most frequently occurs. It thus has a chance of acting vigorously on the young parasites. If 45 grains have to be administered give this quantity in three doses; if 60 grains, in four doses. Where larger doses are indicated 10 grains may be given every 2 or 3 hours as required.

*Method of administration.*—Bates agrees with ROGERS and THAYER who consider the intramuscular injection method inferior to administration *per os*. He finds quinine given in this way is absorbed slowly and sometimes scarcely at all. Despite all aseptic precautions indurations and abscesses may occur. He concludes therefore that injection is not a method of election but of necessity, *i.e.*, it is useful in pernicious cases. So long as the patient can swallow he gives quinine by the mouth. Even when vomiting is troublesome it is well to persevere, repeating the dose and, if necessary, aiding its retention by a small hypodermic injection of morphia, the application of sinapisms to the epigastrium, &c. Although he does not condemn the intravenous method he fights shy of it, having been rendered timid by serious results in a case in which it was employed. He reserves it as a last resort. As regards hypodermoclysis [subcutaneous injection in large dilution] he says that JAMES has now abandoned this method as it causes too much pain and in some cases the temperature persisted in spite of it. Bates himself does not now employ it.

Turning to the question of preventing relapse he advocates a modification of the interrupted method of quinine administration originally introduced by MARCHIAFAVA and BIGNAMI. By this means the tendency to produce strains of the parasite feebly "fast" to quinine is averted. A certain amount of intelligence on the part of the patient is required as will be evident from the following details:—From the last day of the febrile attack count 7 days, note this date and also each seventh day thereafter for 6 or better 8 weeks. On the day before the 7th day take quinine in full doses, usually 30 grains *per diem* and continue this dosage throughout the 7th or "pivotal" day and the day following it [*i.e.*, for three consecutive days]. Discontinue and resume at the next period.

Bates concludes by stating that in his experience the effect of large doses continued over long periods is practically *nil*. He mentions a case where by mistake 30 grains of quinine were taken daily for seven months without any ill effect. Tolerance was speedily established and no harm of any kind resulted.

[This useful paper has been reviewed at some length for it discusses certain questions often ignored by the text-books or on which they give forth an uncertain sound. At the same time all will not agree with the author's views on the value of intramuscular injections. It is curious that he does not mention the salt of quinine which he usually employs, but possibly a discourse upon the merits of different preparations is reserved for a later paper].

A. B.

#### LABORATORY AND EXPERIMENTAL.

THOMSON (John Gordon & David). **The Growth and Sporulation of the Benign and Malignant Tertian Malarial Parasites in the Culture Tube and in the Human Host.** — *Proc. Roy. Soc. Series B.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 77-87. With 1 coloured plate.

A very interesting and well illustrated paper which increases our knowledge both as regards the culture of malarial parasites

and their morphology. It is convenient to give in the first instance the author's summary of their work.

(1) "The malignant tertian parasite has been successfully cultivated after the method of BASS and JOHNS on twelve occasions, and the benign tertian on three

(2) "It is unnecessary to remove the leucocytes from the blood before incubation. The optimum temperature would appear to be about 38° C, and the parasites may grow successfully at a temperature as low as 36° or 37° C

(3) "The time required for the full development of the parasite *in vitro* varies, but this variation is partly due to the age of the parasite at the time of incubation

(4) "The cultures of benign tertian differed from those of malignant tertian in that there was no tendency to clumping of the parasites in the former, either before or during sporulation.

(5) "This difference appears to us to explain in a satisfactory manner why only young forms of malignant tertian are found in the peripheral blood, as the clumping tendency of the larger forms causes them to be arrested in the finer capillaries of the internal organs. It also explains the tendency to pernicious symptoms, such as coma, in malignant tertian malaria. All stages of the benign tertian parasite are found in the peripheral blood and there are seldom pernicious symptoms, because there is no tendency to clumping

(6) The malignant tertian parasite (*P. falciparum*) is capable of producing in maximum segmentation 32 spores. On the other hand benign tertian (*P. vivax*) produces, as a rule during maximum segmentation 16 spores, sometimes more may be produced, but the number is never 32

(7) "The pigment in *P. falciparum* collects into a definite circular, and very compact mass early in the growth of the parasite. On the other hand, during the growth of *P. vivax* the pigment remains scattered in definite granules throughout the body of the parasite till just before segmentation, when it collects into a loose mass of granules in the centre of the full-grown *Plasmodium*

(8) "The morphology of *P. falciparum* and *P. vivax* in the human host is identical with the morphology of these parasites as obtained in the culture tube "

Of the above conclusions those relating to the culture work were reached by the employment of a slight modification of the original technique employed by BASS and JOHNS. It is as follows:—

"10 cc of blood is drawn from a vein and transferred to a sterile test-tube containing a thick wire leading to the bottom of the tube from the cotton wool plug. 1/10 cc of a 50-per-cent solution of glucose is added to this tube, preferably before adding the blood. The blood is defibrinated by gently stirring with the thick wire. Defibrination should be complete in about 5 minutes. The wire with the clot is then removed and the blood is poured into several smaller sterile tubes (about 1-inch column of blood in each). A rubber cap is placed over the cotton wool plugs to prevent evaporation and the tubes are then transferred (standing upright) to an incubator at a temperature of 37° to 41° C. The corpuscles settle in a short time, leaving about half an inch of clear serum at the top. It is apparently unnecessary to remove the leucocytes by centrifugalisation "

In addition to what is stated in the summary the following points may be noted:—

(i) There would appear to be no need to destroy the complement in the serum as is indeed indicated in paragraph number two of the summary.

(ii) The previous administration of quinine may not only influence the rate of growth of the parasites but may inhibit the growth entirely.



(iii) Under very suitable conditions *P. falciparum* can be cultivated in the original test tube through four complete generations without the medium being in any way renewed. This is illustrated by an excellent coloured plate and it is noteworthy that in the later segmentations there were never so many spores produced as in the first, where 32 were found. The authors think that this is easily explained by the conditions in the culture tube becoming gradually more adverse to growth and development.

(iv) There is nothing to show that the actual penetration of a red cell by a merozoite was witnessed.

(v) The authors think that the discrepancies in the accounts of the number of spores found in *P. falciparum* in the human host are due either to the observation of autopsy smears being made at a time when sporulation had not reached its maximum or being conducted on cases where quinine had been given before death.

(vi) As regards the cultivation of *P. vivax* it is important to draw the blood from the patient when the young forms predominate, so that it is then certain that what is seen in the tubes is undoubtedly a further development of the parasite.

(vii) The recent teaching of Ross on segmentation is quoted. It has in part been confirmed by this work and is to the effect that in the case of the benign tertian parasite there are four splits, i.e., 16 spores, with the quartan parasite three splits, i.e., 8 spores and with the malignant parasite five splits, i.e., 32 spores.

[While, as stated, the coloured illustrations are well executed, it must be admitted that Figs. 13, 15, and 17, illustrating young intracorpuseular parasites of the new generations, are not wholly convincing. No fault can be found with the ring form shown in Fig. 20, but, in the light of SCHILLING-TORGAT'S recent work, the other forms illustrated might not be parasites.]

**BASS (Charles C.) & JOHNS (Foster M.). Cultivation of Malarial Plasmodia (*Plasmodium falciparum*) in vitro in the Blood of a Diabetic without the Addition of Dextrose.—*Amer. Jl. Trop. Dis. & Preventive Med.* 1913. September. Vol. 1. No. 3. pp. 246-249.**

In a previous paper (see this *Bulletin* Vol. 7, pp. 22-24) the authors pointed out that in order to cultivate the asexual cycle of the malarial plasmodium *in vitro*, dextrose had to be added to the culture medium. They now mention the case of a diabetic (illness of three years' standing) who had malaria for seventeen days and had been taking quinine irregularly since the first day of fever. In the blood of the patient, without the addition of dextrose, they were able to cultivate *P. falciparum* without difficulty. Controls with non-diabetic blood showed no growth and speedy death of the parasites save, sometimes, in cases when the blood was drawn after a heavy carbohydrate meal, or following violent exertion, conditions which increase its dextrose content. It is possible that any such increase may favour the growth of malarial plasmodia *in vivo*. This could not be determined in the diabetic case as quinine had been taken. At the same time it is perhaps

noteworthy that the fever and the parasites persisted although sufficient quinine had been taken to control an ordinary case of malaria. The clinical history of the patient is given in detail.

A. B.

ZSCHUCKE (Hans). Ueber den Ausfall der Müller-Brendel'schen Modifikation der Wassermann'schen Reaktion bei Malaria.—*Berlin Klin. Wochenschr.* 1913. Sept. 15. Vol. 50. No. 37. pp. 1716-1719.

The fact that a certain proportion of cases of malaria give a positive Wassermann reaction led the author to apply the Müller-Brendel modification of the test to a series of seventeen cases, of which seven were benign tertian, six tropical, one quartan, and three, clinically and otherwise, cases of malaria but without parasites in the peripheral blood. Zschucke modified the very delicate Müller-Brendel method by employing a new control as recommended by PLAUT.

Cases exhibiting partial or delayed haemolysis as well as those in which it was complete were admitted as positives. Out of seventeen cases only three were negative, *i.e.*, a case of benign tertian, a case of chronic tropical malaria and a case of chronic malaria showing no parasites in the blood. In seven of seventeen cases DA ROCHA-LIMA applied the ordinary Wassermann test. Of these no less than six were negative, the single positive case being one with rheumatic complications in which the positive result changed later to a negative reaction.

This tendency towards a weakening and final disappearance of the reaction which is characteristic of the ordinary Wassermann test in malaria is also found with the Müller-Brendel modification. The author discusses this peculiarity and considers fully the various factors which may play a part in bringing it about and also in influencing the occurrence of the test itself. It seems evident that one is much more likely to get a positive result with the Müller-Brendel modification in malaria than with the original Wassermann method. A record of the cases and the results obtained, arranged in tabular form, completes an interesting paper, chiefly however of value to the laboratory worker.

A. B.

DE HAAN (J.). Ueber das Vorkommen der Wassermann'schen Reaktion bei akuten Malariakrankheiten in den Tropen.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1913. Oct. Vol. 17. No. 20. pp. 693-705.

This is the record of a very carefully conducted research. The author enters fully into the literature of the subject and shows that the results hitherto obtained by different authors are not sufficiently in accord to render his research superfluous. The latter was conducted in Java and the blood sera of 163 cases of acute malaria were tested with respect to the presence or absence of a positive Wassermann reaction.

A great difficulty in any such investigation is, of course, the possible presence of a latent syphilitic infection in a malarial patient. In the Tropics the question of yaws has also to be considered.

Of the 163 patients examined 41 were Europeans, 117 Javanese, 3 Chinese, 1 Japanese and 1 Cingalese. In every case the diagnosis of malaria was confirmed by blood examination and the species of the parasite present noted. Further, so far as possible, an accurate enquiry was made into the possibility of previous or concurrent syphilitic infection. Thus, in each case, not only was the venereal history the subject of close enquiry but an examination of the genitalia for scars was made and a search conducted for enlarged lymph glands. Yaws was excluded. The question raised by JAWORSKI and LAPINSKI as to the possibility of the presence of quinine in the blood being a cause of a positive Wassermann reaction was kept in mind and all patients were asked if they had taken quinine recently.

The author's statistics are interesting. Of the 163 cases 61 were benign tertian, 9 quartan, 88 tropical (sub-tertian) and 5 mixed (benign tertian and tropical).

The Wassermann reaction was positive in 63 cases, a percentage of 38.6. It was obtained in 20 tertian cases, 5 quartan, 37 tropical and 1 case of mixed infection. A negative result was forthcoming in 95 instances. In the case of 5 Javanese the serum was found to have strong inhibiting properties.

The positive result was found in 5 Europeans, 51 Javanese, 2 Chinese, 1 Japanese and 1 Cingalese.

De Haan points out how different his results are to those of BLASI who, in Italy, obtained a positive result in 51.6 per cent. of the cases he examined. BLASI's results also differed as regards the reaction in the various kinds of malarial infection.

De Haan thinks that possibly BLASI did not sufficiently exclude previous syphilitic infection. He gives some interesting details as to the information furnished by his own search for evidence of syphilitic infection and in this connection he also instituted enquiries as to other venereal diseases from which his patients might have suffered. Twenty-seven persons were proved to have had venereal trouble. Of these 16 showed scars on the penis but only 8 of the latter gave a positive Wassermann reaction. After excluding every doubtful case de Haan yet found 10 patients in whom the test was positive; out of the 63 positive cases only 12 had taken quinine shortly before their blood was tested. In a praiseworthy endeavour to be yet more certain as regards his results and conclusions the author continued his observations on 30 of the 63 positive cases. He treated them with quinine and after varying periods again subjected them to the Wassermann test. In 11 of the cases a previously positive reaction became negative, apparently as the result of the treatment and amongst these 11 cases all three forms of malaria were represented. The time at which the change takes place seems to vary and perhaps if the investigations had been continued on the 19 cases which remained positive these might also have exhibited the alteration. At any rate the author has conclusively shown that in several persons suffering from one or other form of acute malaria a positive Wassermann reaction was present which was due to the malarial infection. After quinine treatment the reaction became negative but changed again to positive when relapses occurred.

He says that it is not yet clear why all cases of acute malaria do not give a positive reaction but advances the hypothesis that, as the action is in part at least a lipoid one, some malarial infections may cause such changes that a lipoid reaction is present in the blood serum whereas others have not this effect. A lengthy table, embodying the result of this careful research, concludes the paper.

A. B.

**BROWN (Wade H.). The Renal Complications of Hematin Intoxication and their Relation to Malaria.**—*Arch. Internal Med.* 1913. Sept. 15. Vol. 12. No. 3. pp. 315-321.

The author having considered the blood changes produced by alkaline hematin [see this *Bulletin*, Vol. 2, p. 341] now discusses the renal complications of hematin intoxication. This has been studied in rabbits, the hematin and hematin solutions being the same as those used in former experiments. The effects on the urine and on the kidneys are considered separately. The author states that—

"It is not possible to correlate closely the renal complications of hematin intoxication with those of human malaria. The disturbances of function and the lesions of the kidneys have been found much more pronounced in hematin intoxications than in comparable grades of malarial infection in man, while the predominance of the glomerular lesions observed in these experiments is not found in malaria. These differences are partly due to the difference in the concentration of hematin in the blood. It is believed, however, that the analogy is sufficiently close to render the facts disclosed by these experiments of value as a basis for a clearer comprehension of the mode of production of the renal complications of human malaria."

The following is the summary of the work accomplished:—

'1 Mild grades of hematin intoxication produce degenerative lesions in the kidneys and the urine shows a trace of albumin and casts

"2. Severe grades of hematin intoxication result in extensive dilatation, injury and occlusion of the renal vessels by hyaline thrombi or emboli, all of which are most pronounced in the glomerular vessels. Extensive degeneration and necrosis of tubular epithelium, hemorrhages and even anemic infarcts result from these vascular lesions. In such cases, the urine presents the characteristics of an acute nephritis

"3 In rare instances of severe hematin intoxication hemoglobinuria may occur

"4 During the period of recovery from acute hematin poisoning and in chronic poisoning, the kidneys show both degenerative and proliferative processes. The glomerular tufts shrink and the capsular space and tubules are more widely dilated. The tubular epithelium shows degeneration and active regeneration with abundant mitotic figures. There are foci of round-cell infiltration and of connective tissue increase. There is also a slight diffuse increase in connective tissue. The urine is increased in amount and contains albumin with hyaline and granular casts.

"5. The renal complications of hematin intoxication are believed to be due primarily to dilatation, injury and occlusion of renal vessels under the action of hematin."

It will be seen that no light is thrown on the problem of haemoglobinuria though in a small percentage of cases it has been produced by intravenous injection of hematin.

A. B.

ZOLA (L.). *Studi sulla Malaria. Sul Potere Emolitico dello Siero e dei Globuli Rossi e sulla Prova di Bordet e Gengou nella Malaria.* [On the Haemolytic Power of the Serum and the Red Blood Corpuscles, and on the Bordet-Gengou Reaction in Malaria.]—*Malaria e Malat. d. Paesi Caldi.* 1913. June-July. Vol. 4. No. 4. pp. 233-239.

This is a laboratory paper. The author finds that the blood of persons who have suffered from malaria acquires a specific haemolytic power towards the red corpuscles of persons suffering from malaria, provided that they contain plenty of parasites. The demonstration is facilitated by a preliminary treatment of the serum by Rossi's method, which consists in placing the serum for half an hour at a temperature of freezing in contact with washed erythrocytes of the sheep; the serum is then centrifuged and used for the haemolytic test. Control experiments show that this power is not possessed by the serum of patients suffering from other diseases. The test consequently possesses some value as an indicator of latent malarial infection. Three pages of tables are given showing details.

A. B.

#### PREVENTION.

BASS (C. C.). *Eradication of Malaria.*—*Interstate Med. Jl.* 1913. Oct. Vol. 20. No. 10. pp. 921-926.

The author points out how serious a disease is malaria in the southern portion of the United States. It takes toll of thousands of lives annually and, in addition, incapacitates great numbers of workers for days or weeks. Indeed he thinks that it is perhaps the most common and important disease met with in practice amongst southern communities. He believes its almost complete eradication is only a question of the necessary funds, concerted action and education, both lay and professional. Once the importance of a great anti-malarial campaign was fully recognised it is certain that sufficient money for its prosecution would be forthcoming from wealthy philanthropists and other sources. Education through the medium of the public schools is required and also through the press of the country and the agency of corporations and employers of labour, such as railroad companies, plantation managers, &c. More thorough instruction in the case of medical practitioners is required and Bass outlines a scheme for the co-operation of medical schools in malarial districts.

He remarks that:—

"6. A point of very great importance brought out by Dr. THAYER, of the Johns Hopkins Medical School, was that if the mosquito is exposed for any considerable length of time to a temperature below 65° F., plasmodia fail to develop in her and those that have developed die. The season during which the temperature does not drop below 65° F. at any time in the Southern States varies in the different sections. It may be safely stated, therefore, that the length of time during which mosquitoes may serve as hosts for malarial plasmodia does not exceed four to six months, except perhaps in the extreme southern part of the United States.

"7. During the other six to eight months malarial plasmodia are kept alive in human hosts, malaria carriers. These are usually those persons

who have had malaria in some form during the previous warm season. The plasmodia may give rise to active symptoms of malaria or no symptoms may occur, especially during the cool season."

Passing to the questions of prevention and treatment the author points out that until the sexual forms appear in the blood, usually about two weeks after infection has occurred, the patient is not a source of infection to others. Hence early, thorough and successful treatment is all important.

"12. Though quinine will probably not kill sexual malarial plasmodia, it will kill the young plasmodia which they produce, resulting in relapse. Since their life is not more than six weeks, it is only necessary to take quinine properly for this length of time to prevent the development of new plasmodia from them and to allow all the old ones to die out. Unless quinine is taken for this length of time the case is likely to relapse and to be a source of infection to others through the agency of mosquitoes.

"All that is required for the complete eradication of malaria is for everybody who had malaria during a warm season to take the proper amount of quinine on each of two consecutive days in each of six consecutive weeks during the following cool season.

"If this statement could be brought with sufficient authority to the attention of all the people, and if the importance of everybody's co-operating could be emphasized, the writer's belief is that a vast majority of the malaria carriers would follow the advice given."

Two serious obstacles would present themselves in the course of the effort to eradicate the disease along educational lines.

(1) Carelessness of, or disregard of advice by, a few infected persons. For these Bass suggests compulsory segregation as in small-pox, &c.

(2) Importation of cases from abroad. It is possible that some kind of quarantine restrictions may yet be necessary, but a more efficient method would be to carry the education propaganda to the countries providing the infected immigrants.

A. B.

SHAVER (P. J.). **Eradication of Malaria.**—*Texas State Jl. of Med.* 1913. Sept. Vol. 9. No. 5. pp. 161-163.

A general paper containing nothing new. The author, however, quotes the evidence of Dr. EVANS of Chicago before the Drainage Congress to the effect that the extinction of the mosquito would add ten dollars to the value of every acre of land in the malarial infected Southern States of America and would double the crops.

A. B.

HUDLESTON (W. F.). **An Analysis of our Present Position with Regard to the Prevention and Cure of Malarial Infections.**—*Jl. R. Army Med. Corps.* 1913. Sept. Vol. 21. No. 3. pp. 320-338.

After a fairly complete summary of our present knowledge of malaria, more especially as regards prevention and cure, and after a consideration of malaria carriers, the problem of latency, and the occurrence of quinine-resistant strains of the parasite, the author deals with the application of this knowledge to the case of Indian cantonments. In the first place he considers anti-mosquito measures, points out how discouraging have been the results of campaigns in India, and attributes their comparative failure to the peculiar topographical and climatic conditions existing there and to the inadequacy of the funds available. He makes

a suggestion for the thorough testing of this method of control on a small scale, but concludes that anti-mosquito measures alone cannot be expected to protect the population of the average Indian cantonment from malarial infection. He is also of opinion that the efficient use of the mosquito net by soldiers in the aggregate will never, and can never, be attained, and that other methods of mechanical protection cannot be properly carried out. He thinks that everyone with experience will admit that, in India, quinine has failed lamentably both as a prophylactic and as a curative agent and attributes this failure to a lack of proper investigation of its therapeutical action. [According to other evidence it is quite as much due to professional and lay ignorance as regards the proper use of the drug. See paper by BENTLEY in this *Bulletin*, Vol. 2, p. 333.] He suggests the following as profitable lines of research:—

“1. The rate of absorption of the various chemical compounds of quinine in common use.

“2. The chemical condition in which these compounds reach the blood, and the method of their excretion.

“3. The effect of quinine on (a) the blood serum, (b) the red blood cells, and (c) the phagocytes.”

In this connection he states that quinine may possibly interfere with phagocytosis and that its effect on opsonic phagocytosis and spontaneous phagocytosis should be tested. He thinks that if ingestion of quinine is found adversely to affect the opsonic power of the blood on bacteria we might justifiably infer a similar effect as regards the malarial parasite.

The author is in accord with JAMES when he says that comparatively small doses of quinine taken over long periods may tend to produce a quinine-resistant strain of parasite. Referring to the culture work of BASS and FOSTER he considers that it would be interesting to test the results of injections of sub-cultures into the uninfected human being, as he thinks that if infection could thus be produced it would suggest that the sexual forms are forthcoming only when the asexual parasites are threatened with extinction. He also raises the question of the manufacture of a protective vaccine from sub-cultures.

[The author and others interested will find useful information in some of the more recent Italian and German papers on the pharmacology of quinine; notably that by CELLI giving an account of the work of GAGLIO and his pupils and that by JUSTI on quinine administration. See this *Bulletin*, Vol. 2, p. 324 and 330, also the papers by MACGILCHRIST reviewed in this number.]

A. B.

UNTERBERGER (S.). Ueber Malaria bekämpfung. (Nach den Vorträgen auf dem Washingtoner internationalen Hygiene-Kongress im Jahre 1912). [The Control of Malaria.]—*Petersburg. Med. Zeitschr.* 1913. Sept. 15 (28). Vol. 38. No. 18. pp. 221-222.

The author passes in review recent developments in the prevention of malaria, considering more especially the methods of

quinine prophylaxis and mosquito reduction. He deals with the work accomplished in the Canal Zone, Panama, and explains what is to be seen and learned both at the Seamen's Hospital and the *Institut für Schiffs- und Tropenkrankheiten* in Hamburg. He also pays a tribute to the pioneer work of MANSON and ROSS. He contrasts the conditions in Italy and Russia at the present time as regards the distribution of quinine. In Italy with 30,000,000 inhabitants 48,000 kilos of quinine are consumed annually; Russia with its 150,000,000 of people only uses a similar quantity. In Russia over 3,000,000 people suffer annually from malaria. It accounts for nearly half the cases of sickness in the Caucasus and the region of the Black Sea and leads to an extinction of the race. This is brought about by the resulting sterility and the frequency of abortion. The author also attributes to its baleful influence arrested or abnormal bodily development as evidenced by the presence of dwarfs, malformed skulls and faulty genitalia. As a result the percentage of recruits for the army is low in the fever-stricken districts. He mentions the presence of mixed forms of malaria and points out that while salvarsan is valuable in benign tertian infection it fails in the other forms. Finally he puts forward a plea for an energetic anti-malaria campaign in Russia, along the two main lines which elsewhere have yielded such excellent results, *i.e.*, quinine prophylaxis and mosquito destruction.

A. B.

HOROWITZ (Joset). **Bodenassanierung zur Bekämpfung der Malaria in Dalmatien.** [Land Sanitation in the Control of Malaria in Dalmatia.] — *Oesterreichische Sanitätswesen.* 1913. Oct. 2. Vol. 25. No. 40. pp. 1345-1350.

After some geological and historical notes by way of introduction the author explains the conditions favouring the prevalence of malaria in Dalmatia. There is much swamp; there are rivers, fed by mountain streams which overflow their banks; there is low-lying land apt to be flooded; there are lakes which form at certain seasons of the year and then dry up leaving desert stretches covered with rotting vegetation. Within the last ten years suitable engineering works costing large sums of money have effected great improvements both in reclaiming land for cultivation and in diminishing the incidence of malaria. Much, however, still remains to be done. The author describes the different kinds of operations required to meet various local conditions. Harbours on the coast have been improved, as at Nona where also the river Ricina has been controlled. Good results have been obtained in this way. The drainage of extensive swamps is an important problem as is the filling up and reclamation of low-lying lands. The mountainous nature of part of the country renders sanitary engineering costly and difficult.

It is sometimes necessary to drive long galleries through the hills to improve drainage and regulate mountain streams, and in certain places canalisation on an extensive scale is a feature of the work.



[The paper does not possess much of general interest but is useful as showing how large engineering schemes can be made to aid the hygienist and as indicating the great difficulties of anti-malarial work in a rugged and backward country like Dalmatia.]

A. B.

**STRAITS SETTLEMENTS. Return of Malarial Fever, Blackwater Fever, Yellow Fever, Filariasis, and Dengue during the year from 1st January to 31st December, 1912.** [ELLIS (Whitmore).] Received in Colonial Office 7 June, 1913.

During the year 1912 out of a population of 728,635 in the Straits Settlements there were 28,422 registered deaths. Of these 4,303 were attributed to malaria while in addition 4,194 deaths were returned as due to "fever." Blackwater fever caused two deaths.

A report by FINLAYSON on the anti-malarial work possesses some points of general interest. Thus in a shallow, silted-up ditch, draining a swamp near Singapore and overgrown with aquatic vegetation, the larvae of *N. rossi* and *M. sinensis* were found in enormous numbers although three types of larvivorous fish were present in abundance. These were *Haplochilus panchar*, *Hemirhamphus fluviatilis*, and *Ophiocephalus striatus*. Again, there is a note to the effect that WATSON thinks that as *M. sinensis* is a feeble carrier of infection it is probable that *N. karwari* or *N. maculatus* vel *willmori* is responsible for the greater number of endemic cases of malaria. Sometimes when engineering works are being conducted even for anti-malarial purposes springs are laid bare and a flow of water is established which it is difficult to abolish. The only way of dealing with such a condition is to lay a drain of some kind. Any such drain tends to silt up quickly but it is better to lay these drains in any case, and when necessary, dig them up, clean them and reconstruct them.

Some experimental work with Anophelines has been conducted. Imagines of *N. rossi* bred out from larvae were fed on patients showing crescents and a few examined with negative results from the sixth day onwards. The majority rapidly died in captivity. A similar result was obtained in the case of *N. maculatus* and *N. ludlowi*. A few *M. sinensis* survived till the tenth day but showed no zygotes. Neither did some adult *N. rossi* captured in the Telok Blangah area.

A. B.

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BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. *Plasmodium cephalophi*, sp. nov.—*Proc. Roy. Soc. Series B*. 1913. Oct. 1. Vol. B 87. No. B 592. pp. 45-47. With 2 coloured plates.

Malaria of antelopes has not hitherto been described; hence the discovery of a plasmodium in the blood of two young duikers (*Halophus grimmi*) is of considerable interest. Both animals were in captivity and it was thus possible to trace the development of the parasites. Some of the various stages are illustrated in coloured drawings by Lady Bruce. One antelope suffered from

an acute attack of fever—coat staring, nose hot and dry—a few weeks after capture and after being placed in the same enclosure with the other duiker which was about three months old when brought to the station at Kasu Hill (Nyasaland). Some weeks after being taken captive a few parasites were found in the latter's blood and it is probable that it was infected in the wild state and that the duiker showing the acute attack became infected from being brought in contact with it. Both antelopes were first found infected during the height of the dry season (October) and despite a systematic search for adult and larval mosquitoes and the use of traps none of these insects could be found in or near the antelope enclosure. A young reedbuck and a young hartebeest in the same compound remained uninfected. The parasite for which, if it proves a new species, the name of *Plasmodium cephalophi* sp. nov. is suggested, is rather like the human quartan parasite, the gametocytes being circular and the schizonts having from eight to twelve merozoites. Moreover the amoeboid movement is sluggish. It differs however in the marked enlargement and pallor of the red cells and in the fact that the pigment which is pale yellow in colour is collected in a single mass. The pigment is the outcome of an intense and very characteristic concentration of a portion of the affected red cells in the food vacuole of the trophozoite, a point well brought out in the coloured plate. This appearance, together with the absence of Schüffner's dots, the dense blue staining (Giemsa) of the protoplasm of the parasite and the changes in the host cell constitute a unique aspect. Another noteworthy point is the great size of the parasites. Some of the free merozoites measure 4 by 3.5 microns, the exact size of a normal red cell of the antelope, and a full grown schizont before segmentation measures 10 by 10 microns.

Pseudopodia have been seen and also scars in the infected red cells recalling the formation of Maurer's dots. The gametocytes show numerous deeply staining chromatin granules in addition to a faintly stained nucleus. These recall those found in the Haemoflagellata and in this connection the authors state that one duiker was naturally infected with a non-pathogenic trypanosome. The macrogametocytes, well illustrated in one of the plates, are the most persistent forms and, but for the presence of pigment, might be mistaken for faintly stained leucocytes. Some curious anomalous forms of the parasite are also figured.

A. B.

PARHAM (F. W.). Quinin and Tetanus. — *New Orleans Med. & Surg. Jl.* 1913. Oct. Vol. 66. No. 4. pp. 302-309.

An account of a fatal case of tetanus following the hypodermic use of quinine. It occurred twelve years ago but the source of infection remained a mystery. The author considers that the problem has been solved by SEMPLE's experimental work in India. He thinks the patient was probably a tetanus "carrier" and that the quinine injections, which produced small areas of necrosis, provided conditions suitable for the local development of tetanus spores into toxin-producing bacilli. These latent spores, as SEMPLE believes, may have been conveyed by leucocytes to the

favourable nidus either from the patient's ailmentary tract through an abrasion of the mucous membrane or from the site of some old wound.

[There is nothing new in the paper which is chiefly a review of some of the literature on the subject.]

A. B.

**BIREAUD. Idiosyncrasie Quinique caractérisée par des Hémorragies Multiples et Variées.**—*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. p. 454.

A curious case at Hanoi in which quinine idiosyncrasy showed itself by causing haemorrhages from the larynx, gums and tongue. At no time was there any haematuria. The haemorrhages came on about two hours after quinine was taken and also followed a dose given by injection. They ceased when the quinine was stopped and indeed led to the quinine treatment being altogether abandoned.

A. B.

**MALAY STATES. Report from the Institute for Medical Research for the Period October 1st, 1912, to March 31st, 1913.** [FRASER (H.), Director, Institute for Medical Research, Federated Malay States.] Received in Colonial Office June 30, 1913.

Of late years there has been an increase in the number of cases of blackwater fever in the Malay States and a determined effort is now being made to investigate the disease. Short notes on some of the cases encountered are given and it is noteworthy that a considerable proportion of the cases diagnosed clinically as blackwater are in reality not examples of that condition. STANTON furnishes information regarding the Malayan anophelines. He has found that, when they leave the eggs, anopheles larvae of whatever species are very much alike, at least in those features commonly supposed to exhibit constant, specific differences. It is only in the later stages that their distinctive characters are developed. Further the form and arrangement of the clypeal hairs of anopheles larvae vary at the different stages of the larva's life. These changes are described for the larva of *A. albirostris* but apply equally to the larvae of all the six species specially studied. Tables are given showing the principal distinctive characters both of female Malayan anopheles and of their mature larvae.

A. B.

## BLACKWATER FEVER.

**MACGILCHRIST (A. C.). The Haemolytic Action of Quinine and its Salts, with Suggestions regarding the Etiology and Treatment of Blackwater Fever.**—i. *Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, & 20, 1912.* pp. 16-17. (1913. Simla: Govt. Central Branch Press.) ii. *Indian Jl. Med. Research.* 1913. July. Vol. 1. No. 1. pp. 119-166. With text-figs and 9 charts.

i. The author deals in the first place with the haemolytic action of quinine and its salts. He gives a list of all the quinine salts

classified according to their haemolytic power *in vitro*. The acid salts are strongly haemolytic, for example the bi-hydrochloride, bi-sulphate &c. The neutral salts such as the sulphate, hydrochloride and valerianate are slightly haemolytic. Quinine alkaloid or base on the other hand is not only non-haemolytic but actually delays autolysis. Under normal conditions haemolysis *in vivo* by acid mixtures of quinine is prevented by the greater concentration of the medium *i.e.* the blood, the presence of serum, the neutralising effect of the blood plasma and the defensive power of the organism exercised chiefly through the liver. He suggests that in presence of an acid dyscrasia and a disordered liver this defensive power would be lost, and thinks that the hypothesis of an acid dyscrasia plus malaria and quinine explains the incidence and geographical distribution of blackwater fever better than any other theory which has been advanced. This view was strongly criticised in a discussion which followed, but for the arguments advanced against it and the author's reply thereto the original publication must be consulted.

MacGilchrist believes in giving quinine in blackwater so long as it is safeguarded by the administration of an alkali.

ii. This is a somewhat extended edition of the paper just reviewed, the author entering more fully into his subject. For example, the literature on the haemolytic action of quinine is considered as is that on the nature and structure of red blood corpuscles, especially as regards the bio-chemical and bio-physical conditions affecting haemolysis. After a reference to the chemistry of quinine where it is stated that its graphic, structural or constitutional formula is not yet definitely settled, the author gives a full account of his experiments on quinine haemolysis both *in vitro* and *in vivo*. His *in vitro* experiments also included investigations into the alleged haemolytic activity of antipyrin which is so often given along with quinine. He found that it had no haemolytic action and indeed considerably delayed autolysis, thus supporting the later views of KOBERT as expressed in his article on blood-poisons in his text-book on "Intoxications." The supposed anti-haemolytic properties of calcium chloride are shown to be no greater than those of sodium chloride, at least as regards protection against haemolysis by quinine bi-hydrochloride. The *in vivo* experiments were performed upon rabbits, intravenous injections of a combination of quinine sulphate and tartaric acid being employed, a mixture which doubtless contains an acid salt of quinine. The note in the preceding review indicates the conclusions at which the author arrived.

In this second paper he considers the various forms of haemolysis which are now recognised. Thus there is chemical haemolysis (dissolution of the membrane of the red cell) as brought about by the action of acid salts of quinine; and physical haemolysis rendering the membrane permeable to haemoglobin as seen in the action of neutral salts of quinine and in autolysis. These are the true forms of haemolysis described by MOTT. In addition there is haemolysis caused by changes in osmotic tension where a rent may take place in the corpuscular membrane and that produced by strong solutions of phosphoric acid. The latter seems to be an exaggerated physical haemolysis.

The section on blackwater fever has also been somewhat expanded but the author's final views, both on this subject and on quinine haemolysis generally, are perhaps best indicated by his summary which is as follows:—

"1. Acid salts of quinine, *e.g.*, bihydrochloride ( $Q. 2 HCl$ ) and bisulphate ( $Q. H_2SO_4$ ), and double salts of quinine, *e.g.*, quinine-urea chloride ( $Q. HCl CON_2 H_4 HCl$ ) are powerful haemolytic agents. Neutral salts of quinine are only slightly haemolytic, except arsenite and phosphate which have no deleterious action on the blood and arsenate which like quinine alkaloid itself seems to protect the red blood corpuscles and delays autolysis.

"2. A large part of the quinine administered to a man undergoes cleavage in the body and certain derivatives of quinine are known to be strongly haemolytic.

"3. It is generally acknowledged that quinine under certain conditions is capable of precipitating an attack of black-water fever. If, therefore, quinine as such (in contradistinction to a cleavage product) is responsible for an attack of black-water fever, the quinine circulating in the blood is in the form of an acid salt or a double\* salt resembling in constitution quinine-urea chloride.

"4. The results obtained in my experiments on the haemolytic action of quinine and its salts are very suggestive of the existence of a diminished alkalinity of the blood in black-water fever.

"5. This hypothesis of an acid dyscrasia as the third factor (with malaria and quinine) in the causation of black-water fever receives weighty support from an examination of (1) the prophylactic and therapeutic measures advocated in black-water fever, (2) the geographical distribution and seasonal incidence of this disease, (3) the general circumstances under which a person becomes liable to an attack of black-water fever (period of residence, diet, &c.), and (4) the symptoms and signs of black-water fever.

"6. If examination of the blood and urine of black-water fever patients proves the existence of 'acidaemia,' the prophylaxis and treatment of this condition is a simple matter. The action of quinine, like that of salicylates, should be guarded against by the administration of alkalies in all cases where an attack of black-water fever is feared."

A. Balfour.

RICHTER (Geo.). The Etiology of "Blackwater Fever."—*Med. Record*. 1913. Aug. 16. Vol. 84. No. 7. p. 297.

Material derived from the decomposition of "aged" erythrocytes (probably destroyed in the spleen) is converted in the liver into bile pigments. The action of bacteria in the intestines changes these into urobilin (stercobilin) which is in parts re-absorbed and through the portal circulation enters the liver, to be again transformed into bile pigments. These, therefore, have a double source of origin, *i.e.* haemoglobin from the circulating blood and urobilin from the portal circulation. An excess of material from either source or impaired liver function will accordingly lead to urobilinuria. Parenchymatous hepatic diseases may easily remain occult until increased work is thrown on the liver. In malaria the red cells are destroyed by the plasmodia and, as long as the latter are present in the body, there is urobilinuria even when the plasmodia cannot be demonstrated in the peripheral blood.

\* Quinine sulphate is five or six times as soluble in water as is quinine carbonate, but is much more soluble in aerated water than in ordinary water. If quinine circulates in the blood as a double salt, the acid radical of that salt is probably carbonic acid."

Other causes of urobilinuria being excluded, the presence of urobilin means the presence of plasmodia. Indeed no case of malaria can be said to be cured unless urobilin is continuously absent from the urine.

Having pointed out these facts the author concludes that "A coincidence of insufficiency of the liver, perhaps heretofore occult and also due to malarial infection, of malarial blood and eventually of the toxic effect of quinine by its inhibitory effect upon enzymes (bile forming), will have the effect that not only portal urobilin is not converted into the pigments, but that the free haemoglobin in the plasma (derived from destroyed red corpuscles) is not transformed in the liver, but arrives in the kidneys, giving rise to hemoglobinuria, that is to say, blackwater fever."

A case is mentioned where a patient, convalescent from tropical malaria, exhibited not only an enormous amount of urobilin in the urine, but also, after precipitating the urobilin, a very large amount of haemoglobin, though the urine when passed had a normal colour. The blood showed, no parasites but both liver and spleen were much enlarged and very tender on pressure.

A. B.

BOYE. Fièvre Bilieuse Hémoglobinurique. Essai de Traitement par le Sérum Antivenimeux.—*Ann. d'Hyg. et Méd. Colon.* 1913. Apr.-May-June. Vol. 16. No. 2. pp. 447-449.

The author was led to test the efficiency of the anti-venin of CALMETTE in a case of blackwater fever in French Indo-China, owing to the fact that such serum contains anti-haemolytic properties as shown by its action *in vitro*. The case is described at some length and during its course the resistance of the patient's red blood corpuscles to hypotonic salt solution was estimated from time to time. Unfortunately this was not done before the serum injections were commenced, but the latter certainly seemed to increase the resisting power of the patient's erythrocytes.

The dose of the serum was 10 cc. intravenously and the same quantity subcutaneously. It was given the day following the appearances of blackwater symptoms *i.e.* July 27th. A quinine injection was given shortly afterwards. An improvement resulted, especially as regards the quantity and colour of the urine voided, but on July 28th the patient's general state necessitated giving him a hypodermic of isotonic salt solution and caffeine. On July 29th the patient was convalescent, all vomiting having ceased and the urinary secretion being abundant and of normal colour. The author, who has had a large experience of blackwater both in tropical Africa and Tonkin, states that he has never seen the haemoglobinuria vanish so rapidly as in this case. The urine cleared gradually, exhibiting a succession of different shades of colour on its course to normal. Although no definite conclusion can be drawn from a single case the author thinks the method worthy of further trial.

A. B.

## SLEEPING SICKNESS.

RODHAIN (J.), PONS (C.), VANDENBRANDEN (F.), & BEQUAERT (J.).  
 Rapport sur les Travaux de la Mission Scientifique du Katanga  
 (Octobre 1910 à Septembre 1912).—254 pp. With 2 coloured  
 plates and 47 text-figs. Annexe: Carte du Katanga, Répar-  
 tition des Glossines. 1913. Brussels: Hayez, Imprimeur  
 de l'Académie Royale. [Royaume de Belgique, Ministère des  
 Colonies.]

The first portion of the section of this report dealing with trypanosomiasis refers to the distribution and etiology of sleeping sickness along the Congo from Leopoldville to Basoko and in certain portions of the State as far as Kasongo. A summary of the results of examination of the inhabitants for sleeping sickness is given in two tables. There follows an account of the distribution and etiology of the disease in the lower Katanga district and of the prophylactic measures adopted. In the lower Katanga the existence and spread of sleeping sickness appears to be strictly bound up with the presence of *G. palpalis*. On the plateau of Biano *G. morsitans* is found as high as 1,600 metres, whilst *G. palpalis* does not extend beyond 1,140 metres. Cases of sleeping sickness found in villages 1,200 metres above the sea level are imported cases. Reference is made to the work of ROUBAUD, KINGHORN and YORKE, and FISCHER on the influence of climatic conditions on the development of trypanosomes in Glossina. The reasons for the non-infectivity of *G. morsitans* on the upper Katanga may be considered, therefore, to be partly the special biological character of this insect and partly owing to the climatic conditions in which it lives.

The conditions of humidity and temperature necessary for the development of *T. gambiense* in *G. morsitans* certainly obtain in the lower territories north of the Katanga, and the authors succeeded in transmitting the human trypanosome by means of laboratory bred *G. morsitans*. What rôle does this tsetse play in nature in the epidemic of sleeping sickness of the lower Katanga? In the valleys of the Fungwe and the Muanza *G. palpalis* is rare in contradistinction to the gravity of the epidemic, whilst *G. morsitans* is abundant in these more humid valleys. At first the authors were of opinion that the part played by *morsitans* was not negligible, but on further studying the life of *palpalis*, they were able to prove that considerable migration of these insects occurred. As a result of their observations the authors have not been able to convince themselves that *G. morsitans* plays an active part in the transmission of sleeping sickness in the lower Katanga, its rôle in nature appears to be only secondary to that of *G. palpalis*. The explanation of this fact is to be sought in the character and mode of life of *G. morsitans* and in the nature of the virus, *T. gambiense*. *G. morsitans*, a fly of the savannah, lives especially on animals; *T. gambiense* is a parasite adapted to man who constitutes its reservoir *par excellence*. If it is proved that antelope can act as a reservoir of this virus, in nature this eventuality does not appear to be frequently realised. Although in laboratory experiments *G. morsitans* infects as readily

as *G. palpalis*, in nature the former fly only rarely has its first feed on an infected host; the second on the contrary, living by preference on human blood, feeds frequently the first time on an infected host. When it is a question of an organism like *T. rhodesiense*, which is as virulent for man as for animals, *G. morsitans* infects very readily, a considerable proportion of the animals of the savannah acting as reservoirs of the virus. Actually then, in nature, *G. morsitans* does not appear dangerous for man in the lower Katanga where the disease is occasioned by *T. gambiense*.

Regarding prophylaxis the authors state that the most important points are firstly to protect the population against the bites of infected *G. palpalis*, secondly to prevent the flies becoming infected, especially in regions still free from the disease, and thirdly to prevent the introduction of the virus of Rhodesia which could be rapidly disseminated by the numerous *morsitans* which live here in conditions favourable to the spread of the disease. These three propositions are discussed at some length.

The authors deal with the transmission of *T. gambiense* by *G. morsitans*, and with the mechanism of the transmission of trypanosomes by *Glossina*. Most of this work has been published previously (see this *Bulletin* Vol. 1, p. 266 and p. 124). By means of a simple apparatus consisting of a U shaped tube, the short limb of which was slightly enlarged and covered with a membrane consisting of the fresh skin of a mouse, the authors were able to demonstrate that *Glossina morsitans* could take up blood under a slightly negative pressure. The apparatus was filled with citrated blood in such a manner that the level of the liquid in the longer limb was 1 cm. above that of the fluid in the short limb where it was in contact with the under surface of the skin.

The first three flies placed on the membrane engorged themselves so that the height of the column of fluid in the longer limb fell to within 2 mm. of the level of the membrane. Four other tsetses fed rapidly and without the least difficulty, and after their repast the level of the fluid in the longer limb was 6 mm. below that of the membrane.

Hence it is obvious that *G. morsitans* does not require fluid under a positive pressure in order to engorge itself. The authors believe that the reason why tsetses do not feed on extravasated blood, which is not covered by a membrane, is purely mechanical, as the fly in biting is accustomed to pierce a membrane with its proboscis.

The *Glossina* prefer blood to the serum only, with which they will only engorge themselves when very hungry. Neutral red does not appear to damage the flies but sodium emetic, even in very dilute solutions, has a deleterious effect on their digestive functions. Fifteen flies were fed on blood containing sodium emetic (1 part to 20,000 of blood) and eleven died within four days. The blood is more readily taken if warmed to 37° C. than if offered cold. It is important to take aseptic precautions as, when the blood is contaminated with bacteria, these continue to multiply in the gut of the flies and cause death.



The second portion of the report is concerned with the trypanosomiasis of large mammals in the same district. Observations are recorded on the nature, etiology and therapy of the various trypanosomal infections of large domestic stock and game. By way of introduction the authors define briefly the names used by them; thus '*dimorphon-congolense*' refers to a parasite of the short type without free flagellum of which the narrow forms correspond to the stumpy forms of *T. dimorphon* (sensu LAVERAN and MESNIL) and *pecorum* of BRUCE, and the broad and short forms resemble *T. congolense* of BRODEN or *nanum* of BALFOUR. These four species are closely related morphologically and if it be possible to differentiate *T. dimorphon* sensu LAVERAN and MESNIL from the others by means of forms as long as 20-25 $\mu$ , yet it is quite impossible to distinguish morphologically *T. congolense* and *T. nanum*.\* The differential character of the last two lies in the fact that the latter is non-inoculable into small laboratory animals (dog, guineapig and rat); but aflagellar trypanosomes from the dog cannot always be inoculated into the guineapig and rat, so that in Africa where an indefinite number of animals is not available the identification of parasites morphologically closely allied is often very difficult.

The trypanosome of nagana (PLIMMER and BRADFORD) is a parasite which always has a free flagellum and corresponds in its general appearance to *T. evansi*. More recently BRUCE has described the parasite which bears his name as a dimorphic parasite and as such it takes its place in the *gambiense* and *pecaudi* group. The authors designate these dimorphic flagellates as *brucei-pecaudi*; they are closely related in appearance to *gambiense*, but easily separated from this by their biological reactions.

The following is a list of trypanosomes found by the authors in various districts during their tour.

1. In the Kibombo, Kassongo and Kongolo region *T. cazal-boui* and trypanosomes of the *dimorphic-congolense* type were found in domestic stock, whilst these parasites and also *T. ingens* were found in various of the antelope. Regarding the transmitting agents in this district the authors established that *cazal-boui* and *congolense* were transmitted by both *G. palpalis* and *G. morsitans*; the exact rôle of *G. brevipalpis* is still uncertain. The agent responsible for the propagation of *T. ingens* is not known.

2. In the lower Katanga pathogenic trypanosomes belonging to the *dimorphon-congolense* group, *T. cazal-boui* and also trypanosomes of the *brucei-pecaudi* type were found. An account of the morphology and biological reactions of each of these types is given.

A series of therapeutic experiments were undertaken on animals infected with one or other of these types of trypanosomes. A number of goats and sheep infected with trypanosomes of the *dimorphon-congolense* type were treated unsuccessfully with arsenical preparations (arsenophenyglycin and orpiment).

\* According to BRUCE and his colleagues *T. nanum* is a strain of *T. pecorum* which has lost its virulence for monkey, dog, and rat by passage through the goat.

In another series of experiments goats and sheep were treated with sodium emetic. The animals bore well doses of .008 gm. of the drug injected intravenously; the trypanosomes disappeared from the blood, but a relapse occurred after 8 or 10 days.

The authors succeeded in keeping two European dogs alive at Sankisia in the presence of numerous *G. morsitans* by giving sodium emetic at critical times in the course of the infection. One of the animals appeared to have reached a state of relative immunity comparable to that of the wild game. It is to be noted that these animals in contradistinction to goats and sheep were at liberty and exposed to the constant bites of *G. morsitans*. Four other dogs, of which one was native, which only received a single dose of the drug succumbed to the infection. Trypaflavin A. had hardly any action on sheep and goats infected with the trypanosome (*dimorphon-congolense*); on the other hand tryparosan proved more successful and the authors consider that this remedy is the most suitable for infection due to the *dimorphon-congolense* type of trypanosome. Administered by the mouth in doses of .5 gm. per kilogramme of body weight given on two days tryparosan causes the trypanosomes to disappear definitely after 48 hours. This dose is well borne by dogs as well as sheep and goats. Intravenous injections of arsenophenylglycin caused only a temporary disappearance of the trypanosomes.

*T. cazalbouri* was encountered in the blood of sheep, goats and oxen and in various antelope. This trypanosome was found to be sensitive to sodium emetic, a single intravenous injection of .1 gm. being as a rule sufficient to cause sterilisation. Of 9 goats and 1 sheep so treated only one relapsed. Tryparosan acted equally well.

Trypanosomes of the type *brucei-pecaudi* were found in a dog, which had a double infection of this and *T. congolense*, and a goat. A single transmission experiment was conducted with 29 laboratory-bred *G. morsitans*. A single fly became infective on the 18th day and was isolated and examined. It was found to have 'infection totale' of the gut and proboscis. The intestine exhibited a permanent culture of the flagellates and in the hypopharyngeal tube of the proboscis were small trypanosomes of the salivary type; attached to the labium were large numbers of leptomonas forms. These findings are similar to those of ROUBAUD (see this *Bulletin* Vol. 2, p. 245). The authors lay stress on the distribution of the trypanosome in the tsetse as a point of differentiation between *T. pecaudi* and *T. brucei* (*sensu* BRUCE), the latter parasite invading the salivary glands of the fly, whilst the former is limited in its distribution to the gut and proboscis.

Of the non-pathogenic trypanosomes *T. theileri* and *T. ingens* were found in antelope of this district.

3. In the upper Katanga district trypanosomes of the *brucei-pecaudi*, *congolense-dimorphon*, and *cazalbouri* types were found in domestic stock. No examinations of the wild game were made.

In a later chapter of the report the authors record experiments which confirm the work of ROUBAUD (see *Sleeping Sickness Bulletin* Vol. 3, p. 27) and DUKE (see this *Bulletin* Vol. 1, p. 271).

They found that it is impossible to produce sterilisation of the proboscis of tsetse fly infected with *T. cazalboui* by feeding the flies on an animal infected with a trypanocide (emetic of soda).

Records are also given of attempts to capture *Glossina* by means of smearing bird-lime (made with linseed oil) on calico and placing sheets of this on the backs of natives. The results were not very satisfactory.

The report closes with a list and account of the bloodsucking diptera encountered by the members of the Commission. An account is given of the distribution of *G. palpalis*, *G. morsitans* and *G. brevipalpis* and of the pupal habitats of the first-named. The authors did not meet with *G. pallidipes*. In one instance 21 empty pupal cases and 3 living pupae were found in very dry and deeply shaded soil accumulated between the large roots at the base of a tree trunk; from the 3 living pupae 2 *G. palpalis* and 1 *G. morsitans* were hatched. The pupae were found to be practically at the surface of the soil or very slightly buried.

W. Yorke.

DA COSTA (Bernardo F. Bruto). *Sleeping Sickness in the Island of Principe. Sanitation, Statistics, Hospital Services, and Work of Official Conservancy Brigade.* Translated by Lt.-Col. J. A. WYLLIE.—90 pp. With 3 plates. 1913. London: Baillière, Tindall & Cox. [2s. 6d. net.]

The report commences with an account of the history of sleeping sickness in Principe and of the reasons why the disease continued to spread unchecked until 1911.

A medical mission was despatched to Principe in August, 1912. The island was divided into three zones, so that each doctor might work in his own zone and be in touch with the prophylactic measures carried out in each estate. The blood of men and animals was examined in order that those infected might be segregated. In a period of ten months 59 new cases of sleeping sickness were discovered in the first zone (2·8 per cent.), 17 in the second zone (2 per cent.) and 49 in the third zone (4·6 per cent.). Nearly 4,000 persons were examined. There is a strong tendency for the disease to disappear from the island, for in 1908, 23·5 per cent. of the population were infected; whilst to-day, not counting old cases, there are only 3 per cent.† The total number of persons attacked is 361, of whom 50 present every appearance of being cured.

Experiments were undertaken to ascertain the value of atoxyl on animals attacked by trypanosomes. [There seems to be considerable doubt as to the species of trypanosome concerned. In 1907-8 the authors came to the conclusion that "they must belong to one of the species *Pecaudi* or *Dimorphon*," but to-day "they consider that the parasites are probably nothing more than

\* DA COSTA (Bernardo F. Bruto) *Trabalhos sobre a Doença do Sono. Sanitamento, Estatística, Serviços Hospitalares e Brigada Oficial na Ilha do Príncipe.*—78 pp. With 3 plates and 3 folding tables. 1913. Lisbon: Typographia "A Editora Limitada."

† For the Report of the previous Commission see *Sleeping Sickness Bulletin*. Vol. 2. pp. 1-7.

different modalities of the trypanosome DUTTON-CASTELLANI.”] They found that atoxyl was of no value as a prophylactic or remedy, and that it was only of use as a tonic.

An account is given of the prophylactic work done by the planters, which has resulted in a fall of mortality by 50 per cent. on some properties and on others by 70 and 80 per cent. Swamps were drained, timber felled in places where *Glossina* took shelter, the pigs were killed, the secondary growth of scrub jungle was cleared, the flies were caught by means of sticky stuff and the infected domestic animals were slaughtered. Sleeping sickness patients were segregated, and finally injections of atoxyl were given immediately on the bite of the fly.

In February, 1911, an ‘Official Brigade’ was created to aid in the campaign against sleeping sickness. At first this consisted of 40 prisoners of war, but with so small a number it was found impossible to operate profitably on waste lands and on those in native hands. The number of the brigade was increased by means of convicted *serviçaes* and more prisoners of war until it reached a strength of 150 able bodied men. In August, 1912, it was ordered that the Official Brigade should consist of 300 men, but up to now this has not been carried into effect. The author writes “We find it extraordinary that though there is a Native League here, that body should not have impressed its less intelligent members with the necessity for stamping out the sleeping sickness and thus escaping the annihilation of their race. But we only find on the part of the natives of the island a sickly fatalism. The natives instead of maintaining cleanliness, let their properties go to ruin, except for little patches which they used for the growing of manioc and millet.”

The Official Brigade carried out prophylactic work along much the same lines as the planters, clearing the scrub, draining swamps and slaughtering wild pigs. It is well known that the fly takes refuge in shady places, damp and frequented by animals, chiefly pig, which allow it to settle on them in large numbers and carry it about from place to place. The animals, besides being vehicles for the fly, afford it a liberal sustenance. The author has seen as many as 30 *Glossina* gorged with blood, hanging on the dead body of a pig; and the headman of the brigade states that whenever a pig is shot by his men, some *Glossina* are to be found attached to the carcass.

The number of flies caught by the brigade was 110,691; that of pigs killed, 235; lagaias, 251; and stray dogs, 272. To this number of dogs must be added 300 killed in the town area. About 65 square kilometres of land were cleared up by the brigade.

The report closes with an account of the sanitation of the town of Sant Antonio.

W. Y.

BLANCHARD (M.). Variations spontanées de l'Infection sanguine chez quelques Malades du Sommeil.—*Bull. Soc. Path. Exot.* 1913. Oct. Vol. 6. No. 8. pp. 581-583.

MARTIN and DARRÉ have drawn attention to the fact that the trypanosomes do not behave in the same way in all individuals

(see this *Bulletin*, Vol. 1, p. 507). The action of atoxyl in trypanosomiasis may be comparable to that of quinine in malaria and it might suffice to prevent pullulation of the parasites if the interval between consecutive injections of the drug was less long than the cycle of evolution of the parasite. BLANCHARD examined the blood of four cases who had never been treated. The first three were in very bad condition, trypanosomes were present in the cerebro-spinal fluid, and there was somnolence and extreme emaciation. The fourth case was in the first stage of the disease and the diagnosis could only be made by gland puncture. The parasites were sought by centrifugation of 10 cc. of blood. The results are given in the following table:—

Date of Examinations.			Souagregou.	Sangou.	Ténégoro.	Malonga.
July 7	...	...	0			
" 9	...	...	0			
" 11	...	...	+			
" 12	...	...	+++			
" 14	...	...	+++			
" 16	...	...	+++	+++		
" 18	...	...	++	+++		0
" 21	...	...	0	+	++	0
" 23	...	...	0	+	+++	0
" 25	...	...	++	++	+++	0
" 28	...	...	0	++	+	0
" 30	...	...	+	++	+	0
August 1	...	...	++	++	+++	0
" 4	...	...	++	++	++	0
" 6	...	...	0	+++	++	0
" 8	...	...	0	+++	++	0
" 11	...	...	0	+++	+++	0

+ Trypanosomes scanty or very scanty.

++ Trypanosomes not scanty.

+++ Trypanosomes fairly numerous.

It is not possible to generalise from so few cases, but the results show how variable is the intensity of infection of the blood even in patients at the same stage of the disease.

W. Y.

MELLO (Ugo). *Le Trypanosoma gambiense a-t-il une Affinité pour le Testicule?*—*Bull. Soc. Path. Exot.* 1913. Oct. Vol. 6. No. 8. pp. 583-588.

Reference is made to the work of UHLENHUTH and EMMERICH (see this *Bulletin*, Vol. 1, p. 677) which demonstrated that in rabbits inoculated with *T. equiperdum* and *T. gambiense* multiplication of the parasites is much more advanced in the testicle than in the blood. As this fact might be of practical value in the diagnosis of sleeping sickness in man the author undertook experiments with the object of verifying the observations of UHLENHUTH and EMMERICH. The strain used was the *T. gambiense* preserved in LAVERAN's laboratory; it had originally come from a case of sleeping sickness in Uganda. Rats, guinea-pigs and rabbits were injected with the virus intraperitoneally or subcutaneously or into the testicle.

The author found that in rats, whatever be the method of inoculation, the trypanosomes always appeared first in the blood. Only rarely parasites were found as a result of puncturing the testicle except in the case where the injection was made into the testicle. In guinea-pigs trypanosomes appeared rapidly in the testicles only in animals where the inoculation was made directly into these organs. In a monkey inoculated subcutaneously the parasites did not appear in the testicle before the blood. The experiments on rabbits were more confirmatory of UHLENHUTH and EMMERICH's work, as the parasites were usually scanty in the blood and often fairly numerous in the testicles. The author, however, does not believe that this is due to a special affinity of the testicular tissue for *T. gambiense*, but simply to the fact that these organs are rendered oedematous by puncturing and that the trypanosomes multiply frequently more abundantly in serum or oedematous fluids than in the blood.

It does not appear that puncture of the testicle can be utilised for the diagnosis of sleeping sickness.

W. Y.

FLEMING (A. M.). *Trypanosomiasis in Southern Rhodesia*.—*Trans. Soc. Trop. Med. & Hyg.* 1913. July. Vol. 6. No. 8. pp. 298-310. With a map.

This paper gives an account of the facts elicited as a result of the investigations undertaken by the Administration of Southern Rhodesia on the first report of the discovery of human trypanosomiasis in their territory.

*G. morsitans* is the only variety of tsetse found in Southern Rhodesia. The fly areas lie in the northern part of the territory, are small in extent and widely separated. Why the tsetse should be confined within these somewhat arbitrary boundaries, when topographically and climatically there are stretches of country to all appearances similar around them, is unknown. After the outbreak of rinderpest in 1896 the fly belts in Southern Rhodesia underwent an extraordinary shrinkage, whilst in many districts fly disappeared altogether. Since then the fly has in some places shewn a tendency to spread back to its old boundaries.

The scattered nature and limited extent of the fly areas reduce the danger of any serious spread of sleeping sickness in Southern Rhodesia and renders its occurrence of secondary importance, for these areas are only sparsely populated by natives and are remote from European settlements. The removal of persons from any fly infested area would not be difficult should necessity arise. An account of the disease in the Sebungwe district is given (see this *Bulletin*, Vol. 2, p. 36). The author summarises the conclusions reached as a result of this investigation as follows:—

1. That trypanosomiasis in man undoubtedly exists in the Sebungwe district.
2. That the infection is small in relation to the population.
3. That the area of infection would appear to be limited to certain villages along the banks of the Busi river.
4. That whether we are dealing with *Trypanosoma rhodesiense* or *Trypanosoma gambiense*, or some new trypanosome, is still unproved.

5. That though fly-sickness in stock has been known for many years, infection of man would appear to be of comparatively recent origin.

6. That in order to prevent the further spread of trypanosomiasis in man within this area, or the infection of adjacent fly-belts in Southern Rhodesia, the wholesale depopulation of this fly-belt is advisable.

7. That unless steps are taken to prevent the spread of the fly, it will probably, given favourable conditions, spread beyond its present boundaries.

Further work showed that the various strains of trypanosomes obtained from infected men, goats and dogs in this area were identical, and morphologically and clinically allied to *T. rhodesiense*.

The chief problems before the Administration may be said to be (1) The prevention of the spread of trypanosomiasis amongst the native population of the Sebungwe district. The most effective method of checking the disease is removal of the native population beyond the fly infested area. (2) Following depopulation there arises the second and much more complex problem as to whether an attempt should be made to exterminate the fly, or whether the country should be turned into a large game reserve, as has been done with fly areas in Zululand. The author is of opinion that in any scheme for the extermination of fly within the small fly belts of Southern Rhodesia the extermination or driving back of game should be included. He considers that if money can be found, more is to be gained by systematic deforestation from without inwards, by driving of roads through the country, and by breaking up large tracts of ground and the cultivation of sugar, cotton or other products of commercial value, and the consequent eradication of game, and the general civilisation of the country.

In conclusion the author writes that the present policy, which it has been decided to adopt for the suppression of trypanosomiasis in the Sebungwe district, is briefly: To remove all natives from this and the adjacent fly belt on the Umniata to fly free areas, which steps will check further local spread of the disease. It is proposed to withdraw the game laws for the area lying to the east of the Sengwe and west of the Umniata rivers and to throw the country open for shooting. The consequent reduction of game in this area will prove a criterion as to whether in consequence of this the borders of the fly belt can be limited, and possibly fly driven back. [The two propositions in this policy seem to be antagonistic. In any serious scheme devised to deal with the game or fly or for the 'general civilisation' of the country the native population is an essential factor. If the population be removed one of the chief enemies of the game and fly is removed; and in the absence of this enemy these will probably increase and spread. If it be desired to get rid of the game and fly it is obvious that the population must be left in the district. It is equally obvious that systematic deforestation and cultivation cannot be undertaken without the native population. Presumably the Administration consider the question to be a ~~serious~~ one or it would not decide upon so drastic a remedy as the ~~evacuation~~ of the country. Such a policy, however, can hardly be regarded as a satisfactory effort to counteract a danger. The game and fly will probably increase in the absence of man and the

mere removal of the game laws are hardly likely to produce much reduction in the quantity of game in a country devoid of native population.]

W. Y.

**BROWN (Alexander).** Notes of a Case of Sleeping Sickness found on the Hills, Twenty-two Miles north of Serenje, in North Rhodesia.—*Jl. Trop. Med. & Hyg.* 1913. Oct. 1. Vol. 16. No. 19. pp. 302-303.

A clinical account of a case of sleeping sickness in a native is given. The diagnosis was made on July 22nd, 1912. There is some doubt as to where the infection was contracted. The history of the patient, a boy of 18 years, is as follows. He was born in Chimese's village (six miles from Dr. Livingstone's grave); he left his home and went to Serenje in 1908 and was employed by the assistant magistrate, whom he accompanied on at least one journey into the Luangwa Valley. In 1909 he was employed by the Mission and returned to his home as a teacher. In August, 1910, he crossed the Luangwa Valley to Nyasaland and in July, 1911, he made a journey to Broken Hill—a route on which there is no 'fly' until near Broken Hill. In the present year he has been only a few miles from Serenje and not to the author's knowledge in a 'fly' area.

The author writes that a case with such a history occurring at an altitude of 4,800 feet in a fly area calls for some attention. As regards the source of infection there are four possible explanations:—(1) That trypanosomiasis is endemic in the blood of some people. (2) That infection occurred in the Luangwa Valley where, however, he has not been since 1910. (3) That he got it when travelling around his own home in 1909. (4) That he was infected at the mission station near Serenje (20 miles from the nearest 'fly'). In that neighbourhood there are no biting flies except *Stomoxys* which is found in the neighbourhood of cattle kraals. As possible carriers of the infection there are, in addition to *Stomoxys*, bugs, ticks and cockroaches. [It appears possible that infection occurred in July, 1911, when the patient went to Broken Hill, near which place it seems likely that the case described by ELLACOMBE became infected (see *Sleeping Sickness Bulletin*, Vol. 4, p. 185).]

W. Y.

#### RESEARCH IN NYASALAND.

**BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE.** Morphology of Various Strains of the Trypanosome causing Disease in Man in Nyasaland. The Mzimba Strain.—*Proc. Roy. Soc.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 26-35. With 3 plates and 2 charts.

It has been one of the objects of the Commission to determine whether the trypanosome causing human trypanosome disease in Nyasaland is restricted to the game and 'fly' of the Proclaimed Area, or if it extends to the north and south along the 'fly-belt.' If it is found to extend over all the 'fly area' in Nyasaland, then



the disease is probably native to the soil and not an importation from Tanganyika or the Congo. The following are the three opinions or theories at issue:—

"First that the human trypanosome disease of N.E. Rhodesia and Nyasaland is caused by a specific trypanosome, *T. rhodesiense*, that the wild game and 'fly' are heavily infected with it, and that *T. brucei* or Nagana, is absent altogether.

"Second, that the wild game and 'fly' are heavily infected throughout these 'fly-areas' by *T. brucei*, but that at certain places or foci another trypanosome, *T. rhodesiense*, occurs, which is pathogenic to man as well as the other animals. That these two species of trypanosomes are indistinguishable morphologically or by their action on animals, except that one is capable of infecting man and the other not. That the only way to separate them is by inoculating man: if the man reacts it is *T. rhodesiense*, if not, *T. brucei*.

"Third, that *T. brucei* and *T. rhodesiense* are one and the same species of trypanosome, and that wherever wild game and *G. morsitans* are found there also will be found cases of trypanosome disease in man. That the cause of the sparsity of cases in man in these areas is due to the fact that man is more or less refractory to the trypanosome, and that it is only rarely that the 'fly' meets with a susceptible subject. That this is the reason why the cases of Human trypanosome disease in the Luangwa Valley and in Nyasaland do not tend to increase in numbers. The disease remains stationary, as it probably has done during the last thousand years. This is the working hypothesis held at present by the Commission.

"These, then, are the points at issue and it would appear that the only way of solving the problem will be by searching and finding out whether cases of *T. brucei* disease, or Nagana, in man occur wherever *G. morsitans* and this parasite are found together. Already cases have been found on the Rovuma river on the borders of German and Portuguese East Africa, and in the Hartley district south of the Zambesi, in Europeans and natives, who certainly could only have contracted the disease in these widely separated districts."

The trypanosome with which this paper deals came from the blood of a donkey at Mzimba about 100 miles north of the northern border of the Proclaimed Area. A detailed account of the morphology of the parasite is given.

The following are the conclusions:—

"1. The trypanosome of the Mzimba strain is the same species as that occurring in the wild game inhabiting the Proclaimed Area, Nyasaland.

"2. It has already been concluded that this species is *T. brucei* vel *rhodesiense*.

"3. Hence it would appear that wild *G. morsitans* occurring in a district 100 miles north of the Proclaimed Area are infected with the trypanosome which causes the Human trypanosome disease of Nyasaland."

W. Y.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. The Trypanosome causing Disease in Man in Nyasaland. Susceptibility of Animals to the Human Strain.—*Proc. Roy. Soc.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 35-45.

The pathogenicity of the five strains derived from cases of trypanosome disease in man in Nyasaland is compared (see this *Bulletin*, Vol. 1, p. 659). The results are given in tables.

The trypanosome is not as deadly to oxen as is *T. pecorum*. Of animals inoculated only one died, after an illness lasting 134

Four became infected and recovered, while the remaining 13 were refractory. The disease is fatal in goats; 22 were used for experimental purposes and not one of them recovered. The

average duration of the disease was 41·8 days (19-72). One of the animals showed swelling of the face, but none developed opacity of the cornea. The duration of the disease in sheep was 41·1 days (16-87). Among the seven used, oedema of the face was noted as a prominent symptom in three. No opacity of the cornea developed in any of them. One baboon was inoculated without success. Twenty monkeys died on an average in 25·8 days (10-51). The average duration of the disease in 25 dogs was 34·3 days (14-78). In eight of these opacity of the cornea and swelling of the face were present. Seven rabbits died on an average in 27·9 days (15-42). A rabbit suffering from this disease presents exactly the same clinical picture as that seen in rabbits suffering from nagana.\* There is swelling around the eyes, then the face puffs up, there is ulceration round the eyes and nose, and thickening of the ears. Towards the end the eyes are completely closed up, the nose much swollen and both eyes and nose discharge a purulent fluid. Guinea-pigs are more refractory than rabbits and often require to be re-inoculated before they take the disease; 15 were used and died on an average in 66·6 days (13-114). Twenty-one rats were inoculated and the disease lasted on an average 30·3 days (13-93).

Table VI.—The Average Duration, in Days, of the Disease in Various Animals. The letter R means that the animal is refractory, that is, not susceptible to the disease.

Strain.	Ox.	Goat and Sheep.	Baboon.	Monkey.	Dog.	Rabbit.	Guinea-pig.	White Rat.
I.	134	34	R	30	24	42	52	21
II.		59		16	32	33	95	44
III.		31		18	42		62	22
IV.		37		39	33	15	57	49
V.		60		25	42	28	102	32

The conclusions are:—

"1. The trypanosome causing disease in man in Nyasaland is fatal to goats, sheep, dogs, and the smaller laboratory animals, killing them, without exception, in a few weeks. It is less virulent to cattle, many of which evidently escape.

"2. No difference in virulence can be made out in these five Human strains.

"3. It is not satisfactorily proved yet to what species this trypanosome belongs, but the Commission at present leans to the opinion that it is *T. brucei* (PLIMMER and BRADFORD)."

W. Y.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. Trypanosomes of the Domestic Animals in Nyasaland.

I. *Trypanosoma simiac*, sp. nov. Part II. The Susceptibility of Various Animals to *T. simiac*.—*Proc. Roy. Soc.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 48-57.

This paper gives an account of the pathogenicity of *T. simiac* (see *Sleeping Sickness Bulletin*, Vol. 4, p. 350). So far as is known the warthog is the only animal among the wild

\* And dourine, according to published descriptions.—A. G. B.

game of the Proclaimed Area of Nyasaland which harbours it. This trypanosome is very virulent in monkeys and the domestic pig, but harmless to oxen, antelope, dogs and the smaller laboratory animals. The rapidity with which the virulence of *T. simiae* becomes modified is also remarkable. When a cage containing wild *G. morsitans* is placed on a monkey and a goat, both animals take the disease, and the monkey in such an acute form that the average duration of life is only a few days. But if it is attempted to pass *T. simiae* from an infected goat to a healthy monkey by the inoculation of goat's blood, the experiment usually fails, showing that a short sojourn in the blood of the goat has almost nullified the virulence of the parasite for the monkey. This loss of virulence is also seen in *T. pecorum*.

Although *T. simiae* somewhat resembles *T. pecorum* in general appearance the two species differ markedly in their action in animals. Details of the animal experiments are given in tables.

Table IV.—The Average Duration of Life in Various Animals infected by *T. simiae*, Nyasaland. Mixed infections are not included. The duration includes the days of incubation; it dates from the date of infection. The letter R stands for refractory.

—	Ox.	Antelope.	Goat and Sheep.	Pig.	Baboon.	Monkey.	Dog.	Rabbit.	Guinea-pig.	Rat.
Average duration in days.	R	R	46·6	5·3	R	10·8	R	R	R	R
No. of animals employed.	4	5	5	9	3	24	21	10	5	5

Table V.—The Percentages of Recoveries in Various Animals from *T. simiae* infection. This table includes mixed infections.

—	Ox.	Antelope.	Goat and Sheep.	Pig.	Baboon.	Monkey.	Dog.	Rabbit.	Guinea-pig.	Rat.
Percentages ...	R	R	87·5	0·0	R	14·3	R	R	R	R
No. of animals employed.	4	5	32	13	3	35	21	10	5	5

In Nyasaland the carrier of *T. simiae* is *G. morsitans* of which 3·4 per 1,000 were found to be infected. The host or reservoir of the parasite is the warthog. Thirty-three of these animals were examined, and *T. simiae* found in three.

The conclusions are:—

“1. *T. simiae* belongs to the same group as *T. pecorum*, and, like the latter, is erratic in its action on animals.

“2. *T. simiae* affects goats, sheep, pigs, and monkeys. Oxen, antelope, dogs, rabbits, guinea-pigs, and rats are practically immune.

“3. The carrier is *G. morsitans*.

“4. The reservoir of the virus is the warthog.”

W. Y.

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. Trypanosome Diseases of Domestic Animals in Nyasaland. I. *Trypanosoma simiae*, sp. nov. Part III.—*Proc. Roy. Soc.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 58-66. With 3 plates.

This paper gives an account of the development of *T. simiae* in *Glossina morsitans*.

There is a well-marked separate and characteristic mode of development in each of the three main groups of trypanosomes. In the first group—the *T. brucei* group—which includes *T. brucei*, *T. gambiense*, *T. evansi* (?), and *T. equiperdum* (?) the parasites develop—at least in the first two named species—at first through the whole length of the intestinal tract, excluding the proboscis, and eventually reach the salivary glands, where forms resembling those found in the blood of animals are developed and these alone constitute the infective stage.

In the second group—the *T. pecorum* group—which includes *T. pecorum* and *T. simiae* the development takes place in the intestinal tract, including the labial cavity of the proboscis—afterwards the trypanosomes reach the hypopharynx, or termination of the salivary duct in the proboscis. In this group trypanosomes are never found in the salivary glands and no blood forms or infective forms are developed until the hypopharynx is reached.

In the third group—the *T. vivax* group—which includes *T. vivax*, *T. uniforme* and *T. caprae*, the initial stages of the development take place in the labial cavity of the proboscis alone; later the hypopharynx is invaded, where again blood forms are developed which again constitute the only infective forms. There is no invasion of the salivary glands and in addition no development takes place in the intestinal tract. [This classification should be compared with those of DÜKE (see this *Bulletin*, Vol. 2, p. 243) and ROUBAUD (*loc. cit.*, p. 245).]

A brief description of the proboscis of *Glossina* is given. This consists of two tubes: one—the labial cavity—for the passage inwards of blood, made up by the coalition of the labrum and labium, the other for the passage outwards of the salivary secretion—the terminal salivary duct or hypopharynx.

Eight transmission experiments were carried out with *T. simiae* and laboratory bred flies. Two were positive and six negative. There was a great difference between the two positive experiments as regards the time required for the flies to become infective. In the first, 50 days elapsed, in the second only 20. This is due to the different temperatures under which the experiments were carried out—62° F. in the case of the former experiment and 83° F. in the case of the latter. Details of the experiments are given in tables; of 173 flies used in these experiments 10 (5·8 per cent.) became infected with a growth of trypanosomes in the intestines and in the proboscis. Only one fly in 31 (2·7 per cent.) became infective when the flies were kept at ordinary room temperature 62° F., whereas four became infected in 45 (9 per cent.) when the flies were kept at a temperature of 83° F. [These observations support the views expressed by KINGHORN and YORKER in their papers on the influence of meteorological con-

ditions on the development of *T. rhodesiense* in *G. morsitans* (see this *Bulletin*, Vol. I., p. 43 and 126).]

In the two positive experiments out of 76 flies dissected nine infected flies were found. The following table gives the results of dissection of these nine flies.

Table VI.

Expt.	Time, days.	Proboscis.		Proventriculus.	Crop.	Fore-gut.	Mid-gut.	Hind-gut.	Proctodæum.	Salivary glands.
		Labial cavity.	Hypopharynx							
754	37	++		++	—	++	++	—	—	—
754	50	++		++	—	++	++	—	—	—
1847	16-26	—	—	—	—	++	++	—	—	—
1847	30-40	+	+	—	—	++	++	—	—	—
1847	31-41	++	+	++	—	++	++	++	—	—
1847	31-41	++	—	+	—	++	++	++	—	—
1847	31-41	++	+	+	—	++	++	—	—	—
1847	32-42	++	+	+	—	++	++	++	—	—
1847	32-42	—	—	+	—	++	++	—	—	—

In order to examine the contents of the hypopharynx a tube containing one of the infective flies was applied to a large coverslip placed on a man's finger. The hungry fly (starved for 24 hours) at once attempted to feed through the glass and in so doing smeared the surface with saliva. This was immediately fixed and stained.

Four of the seven infected flies in Experiment 1847 contained swarms of trypanosomes in both the labial cavity and the hypopharynx. Those in the latter structure closely resembled the blood forms of *T. simiae*, whilst those growing in the labial cavity resembled *Herpetomonas*, and are peculiar in having their non-flagellated extremity prolonged to a snout-like extension. They were assembled in clusters and attached by their flagellar end to the inner surface of the labrum, their prolonged free extremity moving vigorously in the lumen of the tube. On examination of stained preparations of the saliva of an infective fly typical blood forms of *T. simiae* were seen. Further examination of flies by inducing them to salivate on cover glasses revealed the fact that sometimes long, narrow, intestinal forms of trypanosomes are ejected in large numbers, showing that an infected fly has the power of regurgitating the contents of its proventriculus and intestines forward into the labial cavity and probably into the blood stream of the bitten animal. Possibly in this way the proboscis first becomes infected by intestinal forms of trypanosomes which attach themselves to the labrum and enter the lumen of the hypopharynx. Neither the salivary glands nor the salivary ducts beyond the hypopharynx have ever been found infected with *T. simiae*.

The intestines of infected flies were generally packed full of trypanosomes from proventriculus to midgut or sometimes to hind gut. The most numerous forms are long, slender, ribbon-like, very active trypanosomes. It is impossible to differentiate

one species of trypanosome from another by a study of intestinal forms. *T. brucei*, *T. gambiense*, *T. pecorum* and *T. simiae* all present the same appearance. The paper is illustrated by three plates.

The conclusions are as follows:—

1. "That *T. simiae* can be transmitted from infected to healthy animals by the tsetse fly *G. morsitans*."
2. "That *T. simiae* multiplies in the intestines and in the labial cavity of the proboscis of the 'fly.' Here only developmental forms are found, never infective forms."
3. "That the *T. simiae* growing in the intestines of the 'fly' has no specific characters by which it can be distinguished from other species of pathogenic trypanosomes found in tsetse flies."
4. "That the final stage of the development takes place in the hypopharynx, wherein the infective form of the parasite, similar in shape to the trypanosomes found in the blood of infected animals, is produced."
5. "That the flies do not become infective until about 20 days after their first infected feed."

BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. Trypanosome Diseases of Domestic Animals in Nyasaland. III. *Trypanosoma pecorum*.—*Proc. Roy. Soc.* 1913. Oct. 1. Vol. B 87. No. B 592. pp. 1-26. With 6 charts.

This is one of the most important trypanosome diseases of domestic animals in Central Africa, as it affects them all—horses, cattle, goats, sheep, pigs and dogs. A description is given of the strain from the Mvera cattle, the wild game strain and the wild *Glossina morsitans* strain. The morphology and pathogenicity of the three strains are compared. Morphologically they are identical with one another and with the species found and described in Uganda.

A comparison of the three strains in regard to their virulence towards various animals is given in two tables.

Table XVII.—The Average Duration of the Disease in Various Animals, in days. The letter R means that the animal is refractory, that is, not susceptible to the disease.

Strain.	Donkey.	Ox.	Goat.	Pig.	Monkey.	Dog.	Guinea-pig.	White Rat.
Mvera Cattle ...	87?	103?	64			34	R	R
Wild Game ...			56		142	74	R	30
Wild <i>G. morsitans</i>		140	46	21	117	37	41	37

On the whole it may be concluded that the wild *G. morsitans* strain is the most virulent.

Table XVIII.—The Percentages of Recoveries in Various Animals from the Three Strains. The letter R stands for refractory.

Strain.	Donkey.	Ox.	Goat.	Pig.	Monkey.	Dog.	Guinea-pig.	White Rat.
Mvera Cattle ...	80	37	14			0	R	R
Wild Game ...			17		0	0	R	0
Wild <i>G. morsitans</i>		0	6		0	2	0	0

In Nyasaland the carrier of *T. pecorum* is *G. morsitans*, of which 4·6 per 1,000 were found infected by this species: This is the minimum, but the real proportion of infected flies is probably three or four times as great. An account of the development of the parasite in this tsetse fly is to be given in a future paper. There is also some evidence derived from the outbreak among the Mvera cattle that, given infected animals in a herd, it is possible that Tabanidae, Haematopota, or other biting flies may act as mechanical carriers. The evidence that *Stomoxys* plays a similar rôle is unsatisfactory.

The conclusions are:—

"1. The Mvera Cattle strain, the Wild-game strain and the Wild *G. morsitans* strain belong to the same species of trypanosome, *T. pecorum*.

"2. *T. pecorum*, Nyasaland, is identical with the species found and described in Uganda.

"3. It is an important disease of domestic animals in Nyasaland, being destructive to donkeys, oxen, goats, pigs, and dogs.

"4. Its carrier in this district is *G. morsitans*, about 2 per cent. probably of the local wild flies being naturally infected with *T. pecorum*.

"5. Its reservoir is the wild game inhabiting 'fly-country,' 14·4 per cent. of which were found to be infected with this trypanosome. It is hardly to be doubted that 100 per cent. are, or have been, infected.

"6. It is recommended that if infected animals are found in a herd they should be destroyed or segregated, as there is a danger of biting flies other than the tsetse spreading the disease in the herd by mechanical transmission."\*

W. Y.

#### TREATMENT.

KOPKE (Ayres). Traitement de quelques Cas de Trypanosomiase Humaine par le Salvarsan et le Neosalvarsan.†—*Med. Contemporanea*. 1913. Sept. 14. Vol. 31. No. 37. pp. 289-292.

The author refers to his observation at the Fifteenth International Congress of Medicine at Lisbon in 1906 that it is impossible, by means of atoxyl, to cure those cases of sleeping sickness in which the trypanosomes have invaded the cerebrospinal fluid. He is still of this opinion and considers that from the aspect of prognosis cases of sleeping sickness can be divided into two categories, viz., those in which the trypanosomes have invaded the cerebrospinal fluid and those in which they have not.

A number of sleeping sickness patients with invasion of the cerebrospinal fluid were treated at Lisbon with salvarsan. The doses given were '3 to '6 gm., except in one case aged 10 which received only '1 gm. and '15 gm. The injections were given

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\* In the course of this paper the authors write:—"It is certain that it is only by the detailed study of many strains of each of the African species of trypanosomes that a satisfactory classification of this important group of haematozoa will be arrived at. This work must be done on the spot, where the natural conditions for the normal life of the parasite are present, and where frequent opportunity of passing through the invertebrate host—the tsetse fly—exists. It is absurd to expect to arrive at any classification at all approaching a true one by the study of strains of trypanosomes kept for many years and undergoing many vicissitudes in our European laboratories."

† Communication presented at the XVII. International Congress of Medicine, London, August, 1913

intravenously and repeated at intervals of 8 to 10 days; the total number of injections was generally 3, but two patients received 7 injections. The results are given in a table. Of the 15 patients so treated ten are dead. Of the remaining five one has had two attacks of hemiplegia of the right side with aphasia and will probably die in a short time. In patients who present nervous symptoms the author has not observed after treatment with salvarsan the same amelioration that is obtained with atoxyl.

Subarachnoidal injections of neosalvarsan were given in two cases. The first case presented marked nervous symptoms and trypanosomes were fairly numerous in the cerebrospinal fluid. On January 10, 1913, 16 cc. of cerebrospinal fluid was withdrawn and 10 cc. of a solution (1.5 per 1,000) of neosalvarsan was injected. The injection was followed by pain in the legs, tremors and rise of temperature. The following day there was vomiting, tympanites and constipation. The general condition remained aggravated and the patient died on April 14, 1913. The second case presented less well-marked nervous symptoms. On December 24, 1912, 15 cc. of cerebrospinal fluid was withdrawn and 10 cc. of solution of neosalvarsan injected. There followed symptoms similar, but slighter, to those described above. The patient died on May 7, 1913.

In still another case neosalvarsan was injected intravenously. Doses of .45 gm. were given on November 30 and December 7 and 18, 1912. Eleven days after the first dose pericorneal injection of the right eye appeared, accompanied by visual trouble of the same eye. Examination revealed a retinitis with small haemorrhages around the optic nerve. Psychological symptoms appeared on November 12, 1913, and gradually became accentuated until death which occurred on March 25, 1913. The patient suffered also from syphilis.

W. Y.

HECKENROTH (F.) & BLANCHARD (M.). *Le Neosalvarsan dans le Traitement de la Trypanosomiase humaine.*—*Bull. Soc. Path. Exot.* 1913. Oct. Vol. 6. No. 8. pp. 591-592.

The results are given of treating 25 cases of human trypanosomiasis by a single injection of neosalvarsan. The drug used was the neosalvarsan of EHRLICH and was dissolved in the proportion of .45 gm. in 25 cc. of freshly distilled water. This solution was injected intravenously. The dose administered varied from 1 cgm. to 2.1 cgm. per kilo of body weight. Of the 25 patients treated, 16 in good condition and 9 in moderate or bad condition, four were not re-examined, nine remained free from relapse after an average of  $4\frac{1}{2}$  months, two died 3 months and  $1\frac{1}{2}$  months respectively without relapse, and the remaining ten relapsed after an average of  $2\frac{1}{2}$  months.

Although neosalvarsan caused rapid disappearance of the trypanosomes from the circulation for a certain period it does not appear to act regularly on the trypanosomes contained in the



glands. Numerous active parasites were observed in the gland juice 24 hours, 48 hours and 7 days after doses of 1.7 cgm. per kilo of neosalvarsan.\*

W. Y.

MONFORT (F.). *Essais de Traitement des Trypanosomiasés expérimentales par l'Arsénophénylglycine.*—*Bull. Soc. Path. Exot.* 1913. Oct. Vol. 6. No. 8. pp. 588-590.

Adult mice of 15 to 20 gm. infected with various trypanosomes—*T. gambiense*, *T. rhodesiense*, *T. dimorphon* and *T. congolense*—were treated with arsenophenylglycin. A 2 per cent. solution of the drug was injected intramuscularly; the dose was generally 3 mgm. Mice were found to stand 6 and 9 mgm. without exhibiting signs of intoxication. As a result of these four series of experiments the author concludes that the drug is very active against the two varieties of human trypanosome. The sensibility of *T. dimorphon* to the medicament is equally great, whilst *T. congolense* appeared completely refractory, as even doses of 6 and 9 mgm. only produced a very short sterilisation.

W. Y.

#### BIONOMICS OF GLOSSINA.

FISKE (William F.). *The Bionomics of Glossina; A Review with Hypothetical Conclusions.*—*Bull. Entomol. Research.* 1913. Sept. Vol. 4. Pt. 2. pp. 95-111.

There are certain frequently observed and rather mysterious phenomena associated with the bionomics of the better known species of *Glossina*; such, for example, as the local disparity between the sexes and the apparently arbitrary distribution of the fly. It is difficult, if not impossible to explain these, or at least no explanation has yet been put forward which is wholly in agreement with all that has been recorded of the subject. It appears necessary in consequence to consider some part of the generally accepted bionomic history of these flies as being based on insufficient evidence.

The author attempts in this paper to locate the source of possible error, and to formulate a hypothetical bionomic theory which will explain the curious phenomena mentioned and which is closely in accord with generally accepted theories.

As a result of his review of the subject the author comes to the following conclusions which are presented more as a target for criticism than in the expectation that they will be accepted as a correct exposition of *Glossina* bionomics.

1. "In East Africa *G. palpalis* finds the most favourable conditions for rapid increase in such localities as are represented by certain islands in the Victoria Nyanza and certain spots along the uninhabited lake shore. Here it increases rapidly from generation to generation.

2. "Inferentially, birds or the large reptiles, and not mammals, furnish the most favourable food. It is suggested that the results of laboratory experiments to determine the effect of a diet of reptilian blood do not apply literally to the conditions in the open.

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\* More promising results were obtained by BRODEN, RODHAIN and CORIM (see this *Bulletin*, Vol. 1, p. 129).

3. "The disparity between sexes in the catch seems to be a fair index of the disparity prevailing in the locality.

4. "The superabundant individuals reared under most favourable conditions are disposed of by migration of the females, and the disparity between the sexes is thus to be explained.

5. "These migratory flights of the females are quite distinctly different from the ordinary goings and comings in which both sexes participate. They probably take place during the period following eclosion and fertilisation and preceding full sexual maturity. The general tendency is to pass from more to less favourable breeding grounds.

6. "There is no evidence as to the extent of migratory range. Wide extents of water or of open country probably offer no obstacle to flights of this character.

7. "Through possession of such habits the species would theoretically be enabled to dispense with any other element of 'facultative control' over increase. The failure to discover efficient parasites, pathogenic micro-parasites, specialised predatory foes, or other natural enemies capable of exercising such control, might thus be explained.

8. "In the absence of efficient facultative control, it must be assumed that, in general, localities where males predominate are favourable and where females predominate unfavourable to increase; that the species maintains itself in the latter only through immigration, and that it would become locally extinct were this to cease.

9. "When localities actually favourable to increase are compared with localities actually unfavourable it is not improbable that points of difference will be discerned, which may be of value for economic application. Failure in the past to discover constant points of difference between given localities that are fly-free and fly-infested is not improbably due to the fact that both may be equally unfavourable, but that certain localities are fly-free simply owing to their distance from really favourable localities.

10. "The extent to which these tentative conclusions regarding the bionomics of *G. palpalis* in a certain geographical region will apply to the same species in other regions, or to other species, is wholly problematical. Different species will probably differ bionomically in detail, but the broader general principles of most favoured hosts, of most favourable type of breeding ground, of automatic control over increase through emigration, &c., if substantiated for one species, will probably apply to all nearly related or perhaps all species in the genus."

W. Y.

MOISER (Bernard). Notes on a Few Photographs illustrating the Haunts and Habits of *Glossina tachinoides* in Bornu, Northern Nigeria.—*Bull. Entomol. Research*. 1913. Sept. Vol. 4. Pt. 2. p. 145. With 5 plates.

During the last two years the author has investigated the haunts of *G. tachinoides* in Bornu, Northern Nigeria. These flies are limited to small patches of dense jungle situated along the course of small rivers. They are found in quite localised areas, in close proximity to rivers or marshes where there is water all the year round, and where the ground is covered with tall shady trees, mostly tamarinds, and thick undergrowth of thorns and creepers, with some ebony trees. The possibility that the ebony trees are in some way a factor determining the presence of the flies has often forced itself on the author's attention, for they are invariably to be found in the "belts" and the tsetse flies were never found in any locality devoid of them. The tsetse were most commonly seen resting on the under side of small ebony shoots close to the ground. These shoots have small horizontal branches, devoid of leaves except at the tip, and it is on the under side of these horizontal twigs that the flies come to rest.

A noticeable feature of all the belts examined was the large number of warthog present.

The author writes that he intends to cut down the ebony trees only in a belt to ascertain if this will affect the presence of the flies. The paper is illustrated by ten photographs.

W. Y.

CHALMERS (Albert J.) & KING (Harold H.). *The Distribution of Glossina longipennis* (Corti, 1895).—*Jl. Trop. Med. & Hyg.* 1913. Oct. 15. Vol. 16. No. 20. pp. 320-322. With a map.

This paper gives an account of the geographical distribution of *G. longipennis*. The known area of distribution of this fly extends from about 6° north to 4° south and from about 33° to 47° east. This area includes British East Africa, the south and west of Italian Somaliland, the southern part of Abyssinia, and the south-eastern portion of the Anglo-Egyptian Sudan. The localities in which the fly is found are either desert or semi-desert. When seen in elevated regions it occurs between the rivers and not on their banks. The fact that it is usually a night feeder may explain why it is seldom reported by travellers

W. Y.

NÄGLER (Kurt). *Experimentelle Studien über die Passage von Schizotrypanum cruzi Chagas durch einheimische Tiere. Teil I.* [Experimental studies on the Passage of *Schizotrypanum cruzi* through indigenous animals.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Oct. 4. Vol. 71. No. 2/3. pp. 202-206. With 1 plate.

The first portion of this paper deals with the passage of the trypanosome through small laboratory animals at Berlin. The author succeeded in infecting mice, rats, guinea-pigs, rabbits, dogs and a cat, which subsequently recovered, with *Schizotrypanum cruzi*. Canaries, lizards and frogs were refractory. Hereditary transmission from the parent to the offspring does not occur.

Dealing with the cytological characters of the parasite, the author writes that he considers the male and female forms of CHAGAS to be merely transitional forms. The slender forms are probably young parasites, the broader forms are probably adults and represent the stage before division. In the plate which illustrates the article two dividing forms are figured—the one from the blood of a mouse and the other from the peritoneal fluid examined two hours after death. Contrary, therefore, to previous statements division takes place in the peripheral blood. In well-differentiated Heidenhain preparations a distinct small karyosome is frequently seen. Masses of reserve stuff are sometimes present and may simulate a nucleus.

The statement of CHAGAS that the schizogony of *Schizotrypanum cruzi* in the organism of man and laboratory animals represents the multiplication of sexual forms and is in consequence, a gametogony is left undecided as the author has not

encountered these stages. However, he agrees with CHAGAS that the multiplication of the parasite in the tissues, and in the blood, represents an asexual division which determines the increase of the number of flagellates in the circulation of the infected animal.

Attempts were made to repeat BRUMPT's work on the transmission of the *Schizotrypanum* by the common bed-bug, *Cimex lectularius* (*Sleeping Sickness Bulletin*, Vol. 4, p. 286). The bugs were attached to a silver wire after the manner described by NÖLLER. No development of the parasite was, however, observed in the bugs. The author explains the apparent discrepancy of his results and those of BRUMPT by the fact that his experiments were carried out in the winter when the climate was possibly unsuitable.

W. Y.

BATTAGLIA (Mario). Einige durch Trypanosomiasis Dromedarii erzeugte Läsionen. [Some Lesions produced by Dromedary Trypanosomiasis.] — *Centralbl. f. Bakt.* 1. Abt., Orig. 1913. Oct. 4. Vol. 71. No. 2/3. pp. 182-184.

Reference is made to previous work of the author in which he produced ulcerating granulomata by scarification of the genitals of rabbits with *T. brucei*. On obtaining this new trypanosome of the dromedary (*T. dromedarii*), the author performed similar experiments with it. The prepuce of rabbits was scarified under strict aseptic precautions by means of an injection needle dipped in blood heavily infected with *T. dromedarii*. After four days a hard oedematous patch appeared on the site of scarification; this gradually increased and within eight days a scab formed followed by ulceration and finally a true hard granuloma. In the lesions and oedema of the genitals fully formed parasites were numerous. Oedema of the scrotum and orchitis are rarely seen with *T. brucei*. In dogs and rabbits infected with *T. dromedarii* keratitis was sometimes observed, but this appearance is rarer than in infections with *T. brucei*.

W. Y.

## BOOK REVIEWS.

GOSH (Birendra Nath) & DAS (Jahar Lal). **A Treatise on Hygiene and Public Health with Special Reference to the Tropics.**—xix + 378 pp. 1912. Calcutta: Hilton & Co. [Rs. 3-8 or 5s. net.]

In writing on the subject of "Hygiene and Public Health with special reference to the tropics," the authors, in the present immature state of tropical hygiene, must have been confronted with the question whether advanced sanitary methods of Europe should be described and their principles be applied to the tropics, or conditions in the tropics be detailed and selected remedial measures *ad hoc* be considered in direct relation to them. The former is the method followed by Messrs. B. N. Gosh and J. L. Das; and as the authors make an obvious and laudable effort for conciseness, a natural sequel is that the reader, after perusing various methods applicable in temperate climates, is left with the statement that such and such defects exist in India, without radical remedial measures being indicated. This is remarkably so when dealing with ventilation, cooling and construction of houses, where difficulties as to temperature are dealt with. Again, anthrax amongst animals is frequent, and amongst men occasional, in India; hence, some expansion as to the "why and wherefore" would have seemed legitimate, in the interest of prevention, beyond the statement "it occurs amongst animals grazing on damp soil infected previously with the discharge of diseased animals."

Under water-supply, no room has been found for details of the recent excellent bacteriological work of Major CLEMESHA, I.M.S., and an explanation of the comparative absence of microbes in the middle of the Ganges is given on the theory of HANKIN (of 1894) as to a special "antiseptic" being present. In respect to the Pasteur-Chamberlain filter, the loose statement is offered that the candle should be occasionally cleaned "in hot water or the whole may be boiled." Whilst, as to the Berkefeld filter, it is specifically laid down that the candle should be "sterilized" by boiling every third day. The "four-ghurra" filter is held to be satisfactory; the water in the uppermost "ghurra" is to be "boiled and strained" (!) Under conservancy, it is asserted an adult European male passes about 4 ounces of solid excreta daily "while an Indian passes 8 ounces daily." This is a somewhat misleading statement; but the subsequent context shows it is possible that the latter computation should not have been referred to the Indian male, but should have been quoted as, in the authors' opinion, the average amount per head of an Indian population. Such matters may readily be remedied in future editions without materially increasing the size of the volume, should much of the diffuse material collected on the subject of Meteorology be sacrificed.

On the other hand, there is a practical ring in the treatment of the highly important subject of prevention of malaria; after pointing out the utility of larvicides, protective measures, quinine prophylaxis, &c., they state, "that one of the primary measures for the reduction of malaria is efficient drainage and this is of greater importance in India than elsewhere."

The chapter on diet is carefully written and, in this case, the requirements of the Indian are given due prominence; but there is nothing to indicate, European standards being used to arrive at the calculations, that the comparative weights of European and Indian races have been allowed for.

The authors are to be congratulated on joining the still far too small band of indigenous workers, who perceive that in India, as elsewhere, the wealth and contentment of populations are intimately connected with health conditions.

RODHAIN (J.), PONS (C.), VANDENBRANDEN (F.), & BEQUAERT (J.).  
**Rapport sur les Travaux de la Mission Scientifique du Katanga**  
 (Octobre 1910 à Septembre 1912). [Royaume de Belgique,  
 Ministère des Colonies.]—254 pp. Impl. 8vo. With 2  
 plates and 2 maps. 1913. Brussels: Hayez, Imprimeur de  
 l'Académie Royale. [Price not stated.]

This Report gives an account of the work done by the Scientific Commission which left Belgium in 1910. The personnel included three doctors and an entomologist, M. J. BEQUAERT. The Commission reached Leopoldville in September, and, tarrying at various points on the way, arrived at Bukama in the Katanga in March 1911. A camp was chosen at Sankisia (750 metres) on the road from Bukama to Fundabiabo, near the line of the future railway between Kambove and the Congo. Two members of the Commission worked at the laboratory, while two explored the country from the point of view of tsetse flies and trypanosome infections. The first chapter of the Report (27 pp.) deals with African relapsing fever; chapter 2 (45 pp.) with human trypanosomiasis; chapter 3 (44 pp.) with trypanosomes and trypanosomiasis of large mammals; chapter 4 with *Leptomonas davidi* and non-pathogenic trypanosomes of various insects. Chapter 5 is on Haematozoa from the Belgian Congo; chapter 6 deals with various subjects—tsetse-flies, theileriasis, spirillosis of sheep, helminthology and myiasis (see this *Bulletin*, Vol. 2, p. 530). The last chapter deals with the distribution and biology of blood-sucking Diptera in the Belgian Congo, and includes a list of the Diptera that were collected. A map, which is issued under separate cover, shows the Katanga District on a large scale with the distribution of *Glossina palpalis* and *morsitans*. No scale is given and the meaning of some of the marks on the map is not obvious. Part of this Report has been already summarised in the *Bulletin*; others will be reviewed in future numbers (see page 570). The Report contains much that is of value to the entomologist and medical worker in Africa and is well got up. It includes some observations which have been already published.

A. G. B.

BOSTOCK (Leonard). **Health and Sickness in the Tropics. A Guide for Travellers and Residents in Remote Districts.**—x + 94 pp. 8vo. 1913. London: Simpkin, Marshall, Kent & Co., Ltd. [2s. net.]

This little book may be recommended for those for whom it was written, namely travellers, sportsmen, pioneers and those who reside in remote districts of the tropics beyond the reach of skilled medical assistance. A very good account of the diseases met with in the tropics is given, and equally important matters such as haemorrhages, dislocations, fractures and wounds are also suitably dealt with. As the author says, much may be done to prevent trivial illnesses becoming serious and the use of a vade mecum, such as the above, should help the uninitiated very considerably.

In its pages such an one will also find everything that is required to be known about personal hygiene, food, drinking water, filters, clothing, head gear, boots, houses, huts and tents. A preliminary study of such matters before going abroad may make all the difference between comfort and discomfort, or health and illness, and it cannot be too strongly impressed upon the tyro that he should possess a book of information such as the above and should follow out the principles there laid down.

There are one or two small details however in the work which are not perhaps quite accurate. The statement that the urine in blackwater fever is usually at first bright red in colour is not correct; such a condition would rather suggest hematuria.

Again in describing the diseases specially spread by water ankylostomiasis infection is included. Possibly in several cases this may take place, but undoubtedly the chief manner of infection is by way of the skin from damp infected earth.

It is useful also to look for mosquitoes when one gets into one's net at night, and if any are there, they must be destroyed before the occupant goes to sleep.

The much quoted statement that the *Extractum Cassiae Beareanae* is of value in blackwater fever is given. It is doubtful, however, if this drug exerts any such influence on the disease.

G. C. L.

**DANIELS (C. W.). Tropical Medicine and Hygiene. Part I. Diseases due to Protozoa. (Second Edition.)—xv + 277 pp. Demy. 8vo. With 2 coloured plates and 73 figs. 1913. London: John Bale, Sons & Danielsson, Ltd. [7s. 6d. net.]**

A second edition of Part I of Daniels and Wilkinson's *Tropical Medicine and Hygiene* is now to hand. Amongst other changes it will be noticed that this edition is published under the name of the former author alone, and that, with the index, the work now extends to 277 pages in place of the 264 pages of the first edition. In the preface the author states that an alteration has been made in the arrangement and it is hoped that this will diminish unnecessary repetitions. Amongst the most important of these changes is the omission of the chapter on the general characteristics of the flagellata and the insertion of one on prophylaxis in protozoal diseases as the last chapter of the book. This is a distinct improvement, and as this part of the subject is one of the greatest moment it might even have been extended a little further. There have been no very startling discoveries made in the period of time between the two editions (1909-1913) but still work has been progressive and in the case of some diseases, notably kala azar, has been massive in its amount. The new work on Rhodesian and Nyasaland trypanosomiasis also comes into this time and some of this is duly mentioned. It is practically impossible however to deal satisfactorily with a subject so large as human trypanosomiasis in so short a chapter as 20 pages, though it must be said the author has done his best. The second edition, like its predecessor, will be found to be a useful contribution to the literature of tropical diseases and can safely be recommended to all desirous of studying this quickly growing subject.

G. C. L.

## TROPICAL DISEASES BUREAU.

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## BERIBERI.

ELJKMAN (C.). *Aetiologie und Prophylaxis der Beriberi.*—*Trans. xvin. Internat. Congress of Med., London. 1913. Section xxi. Trop. Med. & Hyg. Part 1. pp. 25-40.*

In a long paper opening the discussion on beriberi at the International Congress of Medicine the author mentions that there are two schools of thought as to the causation of beriberi—supporting on the one hand infection, and on the other dietary defects. He describes his experiments on birds and the production of polyneuritis in them, showing that many other foods besides rice may produce the symptoms, and how the neuritis and emaciation are not dependent on one another. In discussing the poison theory, he states that though the hypothesis of a poison in the rice has been disproved, yet the development of a poison in the intestinal tract during metabolism is still admissible. Experiments by feeding different birds and animals have not given constant results and his own were mostly negative.\* He thinks curative experiments are of more value than prophylactic ones, and he goes on to show how widely distributed in food the active properties are, though—as was first shown by GRIJNS—superheating is able to destroy them. SCHAUMANN's nucleo-phosphorus and activator theories are discussed and dismissed. GRIJNS' theory that FUNK's vitamine is a direct nerve food is, he thinks, a good one, but still is of opinion that the poison theory should not be given up too lightly; it may be a poison that in the last instance affects the nerves, like lead and arsenic—but he does not think that BRADDON's theory of a fungus in uncured rice is tenable.

The author thinks that the protective principle lies not so much in the "silver husk" as in the gluten layer. The curative effects of other foodstuffs as Katjangidjo beans, maize, etc., are then discussed, as well as the fat and nitrogen starvation theories. That the very considerable decrease in the Japanese Navy was principally due to the introduction of food rich in proteid he thinks

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\* See FUNK's reply regarding pigeons and fowls in this number.



is very unlikely, as it has been shown by the experiments of FRASER and STANTON and others, that food containing enough proteid for physiological needs will not protect against beriberi if given with polished rice. He states that it is always a perilous proceeding to alter the national food, polished rice is a product of recent culture and does not make for the health of the people eating it—but it must not be thought that a diet of unpolished rice will not cause beriberi, as many instances are recorded among people who live solely on unpolished rice; it is also difficult to explain the time and place distribution of beriberi on dietary grounds. In conclusion the author states that as yet no specific cause has been demonstrated acting either directly, or only when the resistance of the individual has been lowered by dietary defects; neither have specific antibodies, or antigens from various organs been obtained; and with regard to the “partial hunger” theory it must be noted that a diet poor in vitamines does not always cause beriberi, or a diet rich in vitamines necessarily protect against it.

P. W. Bassett-Smith.

NOCHT (B.). Beriberi.—*Trans. xvii. Internat. Congress of Med., London. 1913. Section xxi. Trop. Med. & Hyg. Part 1. pp. 41-44.*

The author following EIJKMAN stated that he fully agreed with his historical and critical résumé of the recent experiments on beriberi. He pointed out that GRIJNS was the first definitely to suggest that beriberi was due to the lack of some food constituent and he (Nocht) independently instituted experiments in his laboratory with this view, mainly carried out by SCHAUMANN, who recognised that certain elements, particularly phosphorus, were of importance. He stated his belief that the phosphorus question was by no means worked out, and at present the percentage of phosphorus is the standard used by clinicians in prophylaxis and systematic dietary (as applied particularly to rice-eating people). In 1909 he expressed the opinion that different forms of beriberi were due to the want of certain substances x, y, z. Both SCHAUMANN and FUNK have discovered traces of some of these substances, the action and effect of which is probably indirect and more complicated than was at first supposed. The author pointed out that a general degeneration of the nervous system takes place, as in some infectious diseases, and he thought it should be spoken of as an alimentary nerve degeneration and not as polyneuritis; he thought that the influence of weather, fatigue, and intestinal catarrh were only of secondary importance. In his opinion rice, bran, yeast, and similar stuffs were therapeutically of doubtful value, and raw food material was more reliable than a chemically prepared sterilised and isolated vitamine, because a single such vitamine represents only one of the factors, whilst we need several vitamines, such as are found in raw food and the first extracts of raw food, for prevention and cure.

P. W. B.-S

CASPARI (W.) & MOSZKOWSKI (M.). Weiteres zur Beriberifrage. [Further Enquiries about Beriberi.] — *Berlin. Klin. Wochenschr.* 1913. Aug. 18. Vol. 50. No. 33. pp. 1515-1519.

The authors concluded, as a result of practical experience in New Guinea, that beriberi was a disease of metabolism due to feeding on polished rice. As some authorities consider it as an infection, at the suggestion of SCHILLING an experiment on one of the authors was made in Berlin where all possibilities of infection could be excluded, the patient being fed on a diet consisting largely of polished rice. The patient was under treatment for eight months or 230 days. During the first 138, and last 10 days, a complete record of the metabolism was kept; this was divided into a preparatory period and 11 periods of 8-20 days, the diet being altered slightly at each period, but always with a rice basis. The food was largely vegetable; no meat, cheese, or eggs were given. The result was a clinical picture which the authors and others considered to be undoubtedly beriberi of the cardiac type; there were no motor symptoms, but this they attribute to the experiment not being continued long enough owing to the state of the patient, but they say all doubt was cleared up by the fact that a rapid recovery took place on the administration of an extract of rice bran. [The tables are given in full but it is difficult to follow the conclusions derived from them.] They found that there was an enormous loss of albumen which could only be explained as due to toxic action; the phosphorus balance only varied slightly and had no relation to that of the nitrogen, evidencing that in this form of beriberi the bones were not affected; the muscular power fell with the loss of albumen, and throughout the oxygen consumption was diminished.

Their belief that a toxic agent is present was confirmed by an experiment on pigeons. These were fed for months on hens' eggs with a little salt and grape sugar: the birds increased in weight and enjoyed excellent health. To this food was added a sufficient quantity of polished rice, when all the birds after a longer or shorter period developed polyneuritis—the controls remaining healthy; the diseased birds recovered when given the rice bran extract. This they consider conclusive proof of a toxic origin, and they therefore conclude that beriberi is not caused by a lack of something in the food, but by a harmful addition to it—in other words to a poison. They are quite aware that up to the present no poisonous substance has been extracted from rice grains and they do not maintain that the poison must be already formed in the food, but on the contrary there are many indications that it arises in the organism only after a diet consisting chiefly of disease producing food. They hold strongly that for theoretical reasons the deficiency theory of FUNK is not sufficient, and hope that these experiments have established the toxic etiology of beriberi.

P. W. B.-S.

HOPKINS (F. Gowland). *Diseases due to Deficiencies in Diet.*—*Lancet*. 1913. Nov. 8. pp. 1309-1310.

The author, in his introduction to a discussion in the Section of Therapeutics and Pharmacology of the Royal Society of Medicine, reviews the etiology of the deficiency diseases, among which he places beriberi, rickets, scurvy, Barlow's disease and possibly pellagra. The paper is mainly theoretical and controversial, containing no new facts, but is of much interest. He points out that a deficiency disease may in some cases occur even when the diet contains the substances necessary for perfect metabolism, owing to the imperfect assimilation of these; thus when unpolished rice is used, an occasional case of beriberi may occur. Also from experiment it is evident that the substances called vitamins are of various kinds for different diseases, and minute quantities of them are apparently able to prevent the diseased condition. The author also brings forward some old experiments of his own, showing that young rats receiving a sufficient dietary for health always failed to develop if the food had been purified by extraction with alcohol, and if the substances which had been removed by the alcohol were returned, or if minute quantities of milk or fresh tissue extracts were added, the animals grew normally.

P. W. B.-S.

CHAMBERLAIN (Weston P.). *The Character of the Rice which Prevents Beriberi and the Manner in which it is Milled.*—*Amer. Jl. Trop. Diseases & Prev. Med.* 1913. Aug. Vol. 1. No. 2. pp. 121-128.

It is known that when a fowl subsists exclusively on ordinary commercial rice as sold in the United States, it will become paralysed in about 30 days; the nerve degeneration is undoubtedly due to the absence from the diet of some substance essential to nerve nutrition. If a man is fed on diet consisting of the same kind of rice he will develop beriberi after about 90 days, the pathological symptoms of the two conditions being identical. If a mother eating the same kind of rice gives birth to a child and continues to suckle it, the infant will develop the disease called infantile beriberi. The malady is generally fatal if nursing is continued, but the child recovers if fed on canned or fresh milk. If fowls or babies are given extracts of the pericarp, prevention and cure can be effected. These facts make it important that the tropical practitioner should have some knowledge of rice and its preparation. The author therefore describes the structure of the rice grain, which consists of an endosperm composed almost entirely of starch granules and a thin outer portion, the aleurone layer, which contains the albuminous material with most of the fat; outside this is the inner skin or pericarp. When this is removed, more or less of the aleurone layer comes off with it, leaving little more than a lump of starch. The different methods of milling are described. The highly milled rice is deprived of the essential substance necessary to

prevent beriberi. A very small quantity of this neuritis-preventing substance is necessary to maintain health. Similar substances appear to be present in many articles of diet—egg yolk, peas, beans, yeast, etc. This element is essential for nerve nutrition; it is destroyed by a temperature of  $120^{\circ}$  C. and by prolonged boiling; therefore a safe undermilled rice may be rendered beriberi-producing if cooked by steam under pressure. The exact chemical nature of the substance has probably not yet been determined; it is not however a phosphorus compound, but is probably an organic base as claimed by FUNK. The .4 per cent.  $P_2O_5$  index of FRASER and STANTON has been raised by STRONG to .5 per cent. For practical purposes the author believes that after a little experience the safety of a rice can be readily determined by mere inspection of the grain, either in its natural state or after staining with methylene blue or with Gram's iodine solution.

P. W. B.-S.

#### ETIOLOGY.

JENNISSEN (J. A. M. J.). *Over Rijstvoeding en Beri-beri onder het Mijnwerkerscorps te Billiton.* [On Rice-diet and Beriberi among the Mine-workers at Billiton.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1913. Vol. 53. No. 4. pp. 570-583.

Some 14,000 Chinamen are employed in the Billiton tin-mines (Dutch East Indies), and for some time past attention has been directed to the connection between the prevalence of beriberi amongst this body of workers and the character of the rice which forms the principal ingredient of their diet.

In the year 1907-8 there was a failure of the rice-crop in the districts of Southern China from which the mine-workers are principally recruited, and as a result it became necessary to import into those districts large quantities of rice from Siam and Rangoon, all of which is polished. As a consequence there was soon after noticed, at the coolie-inspection station at Tandjong-Pandan in Java, a considerable rise in the number of cases of beriberi detected, together with a general impairment of the physique of the recruits. All the cases that could be detected were sent back to China, but in spite of the vigilance exercised a good many sufferers from the disease managed to get smuggled into the mines, and gave considerable trouble to the authorities afterwards.

When the connection between polished rice and beriberi became a matter of general medical knowledge, it was decided to provide that all rice furnished by contract for the feeding of workers in the mines should be unpolished, whilst at the same time the medical inspection of the workers was made more stringent. The result has been, in the last few years, the almost entire suppression of the disease among the corps of workers at Billiton, numbering some 14,000. The few exceptions have been amongst the wood-cutters and other employees who do not draw rice for their rations, but are paid wages and cater for themselves. The rice which

these people buy in the coolie camps is polished Siam and Rangoon rice, and as a consequence they are from time to time brought to hospital suffering from beriberi.

Analysis shows that the difference in content in  $P_2O_5$  between polished and unpolished rice is very considerable. A sample of unpolished rice furnished to the mine gave by the usual analytical methods 0.89 per cent. of ash, and 0.54 per cent. of  $P_2O_5$ , while a sample of polished Siam rice bought in the Chinese camp gave only 0.45 per cent. of ash and 0.29 of  $P_2O_5$ . The two samples were then submitted to the process of cooking by a camp-cook, and it was found that with the unpolished rice the reduction of  $P_2O_5$  was only from 0.54 to 0.44 per cent., reckoned on the dry grain, while with the Siam rice the reduction was from 0.29 to 0.12 per cent., or a great deal more. Pigeons fed on the cooked unpolished rice remained healthy, whilst pigeons fed on the uncooked Siam rice exhibited very shortly the characteristic signs of paralysis.

The storing of rice, even before polishing, for periods of two to three months also seems to affect unfavourably the content in  $P_2O_5$ , in the climate of Java, owing to the sprouting of the germ; but on this point further investigations are in hand.

P. W. B.S.

HEISER (Victor G.). Further Experiences with Beriberi in the Philippine Islands.—*Amer. Jl. Trop. Diseases & Prev. Med.* 1913. Aug. Vol. 1. No. 2. pp. 119-120.

In support of the hypothesis that beriberi in man is intimately associated with the consumption of decorticated or polished rice as a staple article of diet some very interesting facts are given relating to the Culion Leper Colony. Before the use of unpolished rice was begun the deaths among less than 2,000 persons were about 100 per month, the large majority being due to beriberi. After the unpolished rice was introduced the mortality fell to 15-20 per month and there were no further deaths from beriberi. During the autumn of 1911 there was a great shortage of rice, and the price went up so that large issues of polished rice were supplied. In January 1912 beriberi again became prevalent and the mortality rate soon reached 86 per month. In February the use of unpolished rice was resumed and there was an immediate reduction in the number of new cases, and after April the disease entirely disappeared. The author states that after an absence of two years the disease again made its appearance when polished rice was supplied, and that the issue of rice with a .4 per cent. of  $P_2O_5$  stopped the disease, which reappeared with the issue of a rice with .2 per cent. of  $P_2O_5$ . The author believes that the percentage of  $P_2O_5$  is an excellent index of the value of the rice as a food, but that the identity of the substance in the rice which prevents beriberi has not yet been satisfactorily determined.

P. W. B.S.

FRASER (H.) & STANTON (A. T.). **The Etiology of Beriberi.** [Memoranda.]—*Brit. Med. Jl.* 1913. Oct. 25. p. 1091.

Reference is made to the memoranda of FENTON (E. G.), LINDSEY, and LAIDLAW (see this *Bulletin*, Vol. 2, p. 292), who drew attention to the possibility of other factors than rice being concerned in the causation of beriberi. The authors point out that, though it is certain that in the Far East rice is the cause of the disease as shown by actual practice, in other countries probably many other diseases are described as beriberi which are very different and have other etiological characters, and it is not likely that any single factor will explain their causation. They deplore the tendency of most writers to exalt the importance of Polyneuritis gallinarum and to ignore the human disease.

P. W. B.-S.

FRASER (H.). **Report from the Institute for Medical Research [Kuala Lumpur, Federated Malay States] for the period October 1st, 1912, to March 31st, 1913.**—Received in Colonial Office June 30, 1913.

In this report it is noted that information has been received from Dr. WOLLASTON and Mr. KLOSS who were exploring in New Guinea, that in the last expedition, of over seven months duration, the 240 coolies employed had remained quite free from beriberi; this they attribute to the fact that they were supplied with unpolished rice only—as on previous expeditions when polished rice was used the men were decimated by the disease.

P. W. B.-S.

GIMLETTE (John D.). **Beriberi in Kelantan. An Appendix to the Annual Report, Malay States, for 1912.**—Received in Colonial Office, Sept. 27, 1913.

The intermittent presence and absence of beriberi in the Malay State of Kelantan are shown in a series of statistics, and appear to be definitely dependent upon the consumption of polished or unpolished rice. According to the author the disease is particularly frequent when the rice used has been stored a long time and has become mouldy. He states that in Kelantan the rice spoils within three or four days if it is allowed to get damp and is neglected; for this reason the Malays do not store their rice but prefer to pound their paddy in small quantities at a time, thus having it always dry and clean for use. In conclusion he considers, from a review of the history of beriberi in Kelantan during the last ten years, that rice only acts as a vehicle of infection among human beings and not as a causative agent.

P. W. B.-S.

STANLEY (A.). *The Etiology of Beriberi.* [Memoranda.]—*Brit. Med. Jl.* 1913. Nov. 1. p. 1160.

The author, who has had over fifteen years' experience at Shanghai and other parts of China, draws attention to the fact that in gaols, convents and schools, where large numbers live together, the number of beriberi cases is greater than in the surrounding general population, there being practically no difference in the rice used. He states that isolation of the sick and ordinary disinfection of clothing, &c., have little or no effect on an outbreak of beriberi, whereas after destruction of all body vermin and bugs, &c., there are strong reasons for thinking that the prevention of the spread of the disease is immediate and effective, which points to infection by external animal parasites.

[No definite facts are given in proof of this infective theory].

P. W. B.-S.

CASEY (J. P.). *The Etiology of Beriberi.* [Memoranda.]—*Brit. Med. Jl.* 1913. Oct. 25. p. 1091.

The author describes shortly an outbreak of beriberi which affected 60 per cent. of the Boer prisoners at the war camp at Deadwood, St. Helena in 1901-2. It was discovered that the land on which the prisoners' tents were pitched had in 1832 been a camping ground for Indian coolies and negroes, and it is suggested that the infection had come from the ground, the micro-organism having been dormant there all these years. When the old ground was condemned and the camp moved there was a marked improvement in the general health, practically all the cases recovering. No case of beriberi occurred among the troops, some of whom were quartered not far from the original prisoners of war camp.

[In the absence of any details of the hygienic and food conditions of the camps and with the very long period of supposed latent infection, the suggestion does not appear to be of much value].

P. W. B.-S.

#### VITAMINES.

VEDDER (E. B.) & WILLIAMS (R. R.). *Concerning the Beriberi-Preventing Substances or Vitamines contained in Rice Polishings. A Sixth Contribution to the Etiology of Beriberi.*—*Philippine Jl. of Science.* Sec. B., Trop. Med. 1913. June. Vol. 8. No. 3. pp. 175-195.

The authors report a large number of further experiments to determine the composition, preparation and methods for isolating the protective substances against beriberi from rice polishings, and to obtain evidence as to whether there was experimental proof to support BRADDOCK's statement that beriberi only occurred where rice used for food had been stored in a damp place, and that when care was taken to store the rice in a dry place, there was

no beriberi. After an initial series in which fowls were used, three human cases were treated, and as a natural result of the great success obtained by the treatment of cases of infantile beriberi with the extract prepared in the Philippines, this was employed on adults. The results of their work are summarised in the "conclusions," which are here given in full.

"1. Undermilled rice may be stored for one year in a damp place without losing its protective powers against polyneuritis gallinarum. It is improbable therefore that a rice which originally affords protection against beriberi will lose this property by storage even in damp places."

"2. The neuritis-preventing substances or vitamins contained in rice polishings are only slightly soluble in cold 95 per cent. alcohol, since three successive extractions, using a total of 6 liters of alcohol to each kilogram of polishings, fail to remove all of the neuritis-preventing substances from rice polishings.

"3. Strongly alkaline reagents, such as sodium hydroxide, ammonia, and barium hydroxide, destroy the neuritis-preventing vitamin in its free or unhydrolyzed state, and the use of these reagents must be avoided in endeavouring to isolate this substance.

"4. Basic lead acetate does not precipitate the neuritis-preventing vitamin, and a considerable portion of this substance may be recovered from the filtrate.

"5. The therapeutic properties of an alcoholic extract of rice polishings are greatly altered by hydrolysis (treatment with 5 per cent. hydrochloric or sulphuric acid). The unhydrolyzed extract is not poisonous and is only slowly curative. The hydrolyzed extract is exceedingly poisonous in large doses and promptly curative in small doses.

"6. We have confirmed FUNK's observations by isolating a crystalline base from an extract of rice polishings by FUNK's method. This base in doses of 30 milligrams promptly cured fowls suffering from polyneuritis gallinarum.

"7. FUNK's base or vitamin is present in rice polishings in considerable amounts, and only a very small portion of it can be obtained by FUNK's method.

"(1) Because the polishings themselves are incompletely extracted.

"(2) The greater part of this base is lost during the chemical manipulations required by FUNK's method as shown by the facts:

"(a) The curative action of this base, isolated, is from twenty-five to fifty times weaker than the curative action of the original hydrolyzed extract.

"(b) When fowls are fed on polished rice and given a daily dose of this base in amounts corresponding to 10 cubic centimeters of the original extract, these fowls are not protected. Ten cubic centimeters of the original extract or 10 grams of polishings daily are amply sufficient fully to protect fowls.

"(8) Because FUNK's method depends upon the use of barium hydroxide, and we have shown that this reagent destroys this base.

"8. Two groups of substances (purine bases, choline-like bases) may be isolated from rice polishings in addition to FUNK's base and are capable of partly or wholly protecting fowls fed on polished rice against polyneuritis gallinarum, but are incapable of curing fowls that have already developed the disease. The chemical nature of these two groups of bases requires further investigation.

"9. We have confirmed the observation of SUZUKI, SRIMAMURA, and ODAKE, that FUNK's base may be precipitated from unhydrolyzed extract by tannic acid, but did not succeed in obtaining large amounts of this substance by this method.

"10. It is probable that this base or vitamin exists in food as a pyrimidine base combined as a constituent of nucleic acid, but that it is not present in the nucleins or nucleic acids that have been isolated by processes involving the use of alkalis or heat.



"11. The administration of unhydrolyzed extract of rice polishings to cases of adult wet beriberi, or to cases suffering from acute cardiac insufficiency, results in the prompt dissipation of oedema and relief of the cardiac symptoms.

"12. The administration of unhydrolyzed extract of rice polishings to cases of dry beriberi is followed by little or no improvement in the paralytic symptoms.

"13. The administration of FUNK's base to cases of dry beriberi is followed by an immediate improvement in the paralytic symptoms. This should remove the last doubt that dry beriberi is caused by the deficiency of this substance in the diet. It also finally proves that dry beriberi of man and polyneuritis gallinarum are essentially the same disease.

"14. We have succeeded in curing a case of infantile beriberi (of the wet type) by administering that portion of the extract of rice polishings represented by the filtrate from the phosphotungstic precipitate. Since this filtrate does not contain FUNK's base, this is evidence that wet beriberi is cured by some other substance.

"15. Conclusions 11, 12, 13, and 14 are striking confirmatory evidence for the hypothesis previously stated by VEDDER and CLARK that wet beriberi and dry beriberi are two distinct conditions, each being caused by the deficiency of a separate vitamine."

[Clinically many authorities do not recognise any distinction between wet and dry beriberi—the one may rapidly pass into the other.]

P. W. B.-S.

FUNK (Casimir). *Fortschritte der experimentellen Beriberiforschung in den Jahren 1911-1913*. [Advances in Experimental Beriberi Research in the Years 1911-1913.]—*München. Med. Wochenschr.* 1913. Sept. 9. Vol. 60. No. 36. pp. 1997-1999.

Previous investigations had shown that an active principle was present in ordinary rice which was absent in polished rice. From its high power of resistance to acids the author concluded that the substance or substances must contain nitrogen. He proceeded to try to isolate these by the usual methods for N. compounds, testing every substance obtained by means of pigeons. He found that the active principle was present in the precipitate by silver nitrate and baryta, etc. The yield by this method was too small for much work, so he tried another method deduced by the suggested resemblance of the substances to the pyrimidin bases. He tested a large number of the substances so obtained on pigeons and obtained only partial curative results, which however suggested further enquiry. He concluded that the active substances must belong to a new group which he called vitamines. These substances contain nitrogen in a combination which has not yet been found in [? animal] nature, and which animals are not in a position to build up and must therefore draw already made from the vegetable kingdom.

SUZUKI and others had meanwhile isolated nicotinic acid for the first time from rice bran, and also vitamine as a picrate; the latter FUNK so far has not been able to verify. MOORE and others have verified the presence of vitamine in yeast, and COOPER in *Sch.* From his experiments he determined that vitamine is a very unstable body. By using large quantities of the vitamine-fraction he isolated a crystalline substance which was eventually

split up into three chemically pure substances, one of which was nicotinic acid. These individually had little curative effect, but by combining 1 and 3, marked curative results followed; he also found that the earlier precipitates contained the vitamins. He discovered that there was a loss of activity in the pure substances as compared with the extracts; this was accompanied by a loss of a colour reaction to certain reagents as described by FOLIN and MACALLUM. This colour reaction is only produced by the uric acid group, tyrosin, and some phenols, in addition to certain purin derivatives. All food substances which contain vitamins have so far without exception given these reactions, as well as all vitamin fractions. The method for isolation of these unstable compounds without decomposition has at last been discovered and will be communicated shortly.

The author states that we are still in the dark as to the real nature of beriberi, but he still holds the view that the primary cause is the lack of vitamins in the food, and that it is probable that the nerve degeneration is produced in another as yet unknown manner. The extraordinary rapid action of vitamin can hardly be regarded as that of an anti-toxin and it is certain that the nerve degeneration is secondary, as after cure it is still demonstrable histologically, while functionally the nerve action is normal. He classes with beriberi as "deficiency diseases" scurvy, Barlow's disease, and probably pellagra, and rickets. He states that this study has been most valuable in discovering the complex nature of foodstuffs, and the isolation of the active principles in them will increase our knowledge of cell functions. It is now necessary to study the chemistry of the minute constituents of food which demonstrate themselves by their physiological action.

In a note in reply to EIJKMAN'S statement that the results obtained from the study of pigeons cannot be accepted as true of fowls, he states that during the years of his experimental work no difference has been found in the behaviour of fowls and pigeons, either in the inception of the disease or its cure; also that the statement of BARSICKOW that cerolin (alcoholic extract of yeast) has no action on beriberi pigeons has been proved to be wrong.

P. W. B.-S.

**FUNK (Casimir).** *Über die physiologische Bedeutung gewisser bisher unbekannter Nahrungsbestandteile, der Vitamine.* [The Physiological Meaning of Certain Hitherto Unknown Food Ingredients, the Vitamines.]—*Ergebnisse der Physiologie*. 1913. Vol. 13. pp. 126-205 and 547-548. With 6 text-figs. and 1 plate.

In this monograph the author has elaborated his own views with reference to beriberi, together with those of others put forward in many important papers recently published. He not only discusses the food question as bearing on beriberi, but also shows how other groups of diseases such as scurvy, rickets, and

pellagra are dependent upon a deficiency in the supply of food containing certain necessary constituents. Most of the points have been put forward previously, and at the end of each section an excellent bibliography is given. After a short introduction, a description of beriberi and polyneuritis of fowls is given, illustrated by the well known examples of paralysed birds and sections of rice grains taken from FRASER and STANTON'S work. He goes on to discuss the chemical nature of the curative substance called by him vitamine, obtained from rice bran, yeast, and other fresh food substances, its chemical composition and physiological action. The proportional activities of different food substances due to the presence of the anti-neuritic substance are also described (egg yolk and yeast having the highest values). The form in which vitamine is present in nature is mentioned and six pages are devoted to an attempt to explain the nature of beriberi. A table is given of the composition and value of the various purin, pyrimidin, and nuclein derivatives, with their allied forms of allantoin and hydrantoin. The purins have practically no curative action, the pyrimidins more, while that of allantoin and hydrantoin is very marked. He finds that those substances were the most active which in composition most nearly resemble vitamine, and had a nuclein basis; finally that the vitamine fraction from yeast is made up of three substances, one with the formula of  $C_{24}H_{19}O_9N_5$ , another with that of  $C_{29}H_{23}O_9N_5$ , and nicotinic acid. The first with the third, in doses of 1 m.g. injected subcutaneously produced a cure in affected pigeons. From the vitamine fraction of rice bran two substances have been isolated,  $C_{28}H_{20}O_9N_1$  and nicotinic acid. All the derivatives that are obtained from the mother substance are important, but the nicotinic acid is the most active. Ship beriberi is placed with the scurvy group, which differs from tropical beriberi in that the neuritic symptoms are less marked and recovery is more rapid when fresh provisions are supplied. In conclusion he states that these facts open up a new sphere for clinical and physiological research, and that the principles of dietetics must undergo revision and reform. New questions arise as to the composition and preparation of our food, but it is of the utmost importance first to know the relative vitamine content of the various food stuffs.

[Those interested in the subject should read the paper in its original form.]

P. W. B.-S.

WISE (K. S.) & MINETT (E. P.). *Epidemic Dropsy.—Report of Tropical Disease Research in the Government Bacteriological Laboratory, British Guiana, for Six Months Oct., 1912, to March, 1913.* Report received in Colonial Office, Sept. 30, 1913.

In 1912 an expedition of 190 creole mulattoes and black men started from Georgetown for a camp up the Essequibo River, 30 miles from the Brazilian border. The situation of the camp

appears to have been well chosen and healthy; no cases of sickness appeared until four months after arrival, when an epidemic broke out, characterised by pyrexia, general progressive oedema and vomiting. The cases occurred over a period of two months. Out of the 190 men 24 were attacked, 7 of whom died. There was no history of an abnormal number of biting insects, and the food though monotonous appears to have been good, the general labourers having brown creole rice (unpolished) with flour, tinned beef, and tinned fish, etc. The symptoms were those of epidemic dropsy though it is noted that on their return to Georgetown the patients showed signs of oedema and cardiac distress on exertion, and a small amount of albumin was present in the urine of two cases; knee-jerks were present and the men appeared otherwise well. The authors consider the disease as one of mal-nutrition coming under the heading of deficiency diseases as described by FUNK. A similar outbreak occurred during the construction of the Madeira-Mamore railway in the state of Amazonas and Matto Grosso, Brazil, described by LOVEFACE as beriberi. (See below and this *Bulletin*, Vol. 1, p. 484.)

P. W. B.-S.

LOVEFACE (C.). *Peripheral Neuritis in the Amazon Valley.—Texas State Jl. of Med.* 1913. July. Vol. 9. No. 3. pp. 94-95.

In the *Journal of the American Medical Association*, 1912, December 14, the author described the results of his observations on 963 cases of a beriberi-like disease which was present in the Amazon Valley (see this *Bulletin*, Vol. 1, p. 484). In this paper he again discusses the etiology of this disease, and concludes that though beriberi in the east is due to a deficiency condition of the food, in this instance defective diet and consumption of rice could be positively excluded. He considers that it is highly probable that the term beriberi is one that has been used to cover a group of diseases, more or less indistinguishable clinically, and that the question of the etiology of beriberi cannot yet be regarded as settled. [No new facts are given.]

P. W. B.-S.

KARIYA & TSUJUKI. *The Blood Pressure of Beri-beri Patients.—Sei-I-Kwai Med. Jl.* 1913. Aug. 10. Vol. 32. No. 8. pp. 109-110. [The original in No. 10, Vol. 27, 1913, of *Jl. Tokyo Med. Assoc.*]

The authors from a study of 28 cases of beriberi, free from kidney complications, etc., found that the maximum blood pressure of the radial artery was less than that of healthy people. This change is most marked in the cardiac and severe types of beriberi, and the blood pressure was inversely proportional to the state of the disease. It is therefore of use in diagnosis and for prognosis.

P. W. B.-S.

## POLYNEURITIS OF BIRDS.

WELLMAN (C.) & BASS (C. C.). *Polyneuritis Gallinarum caused by Different Foodstuffs. With Special Reference to the Effect of Commercial Rice Coating on Neuritis Production.*—*Amer. Jl. Trop. Diseases & Prev. Med.* 1913. Aug. Vol. 1. No. 2. pp. 129-139. With 3 plates.

Certain experiments are described carried out to determine what common articles of diet besides rice would give rise to polyneuritis in fowls when used as the only food, and whether the commercial samples of polished rice coated with glucose and talc, locally milled, would also produce the disease in fowls. The process of thus preparing the grain is described; the coating gives the rice a highly polished appearance, and makes it keep better, but the amount of glucose and talc varies greatly in different samples—the average amount of glucose equals about one part by weight in eight hundred parts of the finished article; the quantity of the talc is about half this. The following conclusions are given:—

"1. Glucose and talc, when fed together or separately, do not produce *Polyneuritis gallinarum* in birds receiving a diet which does not itself produce the disease.

"2. Glucose and talc in large amounts do not prevent prompt recovery from *Polyneuritis gallinarum*.

"3. *Polyneuritis gallinarum* can be produced by feeding milled rice and many other food substances which have neither glucose nor talc on them.

"4. Glucose and talc, therefore play no part in the production of *Polyneuritis gallinarum* which results from an exclusive diet of 'polished' rice.

"5. These experiments indicate that legislation or regulations against the sale of 'polished' rice, based upon the fact that *Polyneuritis gallinarum* results from feeding it as an exclusive diet, are not warranted.

"6. The evidence here presented indicates that several other common articles of diet produce *Polyneuritis gallinarum* as certainly as does rice, either 'polished' or unpolished: in three instances these other foods produced the disease in quicker time than did rice.

"7. There is, therefore, more argument against the sale of these common articles of diet, sago, Irish potatoes and corn starch, than there is against the sale of rice, 'polished' or 'unpolished,' milled or undermilled."

P. W. B.-S.

WELLMAN (C.). *On the Production of a Beriberiform Polyneuritis in Fowls with Substances other than Rice.*—*Southern Med. Jl.* 1913. Aug. Vol. 6. No. 8. pp. 516-518. With 9 text-figs.

This short paper was apparently written to point out again that polyneuritis of fowls is not alone produced by the use of polished rice, but can be brought about by feeding on other carbo-hydrates, as cane sugar and corn starch. The author also found that a mixed diet with small doses of oxalic acid produced a similar paralytic condition, as shown in the birds depicted in the illustrations. It is possible that the excessive use of cassava starch in Brazil, and the inordinate drinking of cane juice by the negroes in the Antilles, may have something to do with the outbreaks of beriberi which are reported from these countries.

P. W. B.-S.

VOEGTLIN (C.) & TOWLES (C.). **The Treatment of Experimental Beriberi with Extracts of Spinal Cord.** — *Jl. of Pharmacology & Experim. Therapeutics.* 1913. Sept. Vol. 5. No. 1. pp. 67-76.

After giving a resumé of recent experiments on the curative substances obtained from rice, bran etc., the authors describe their results of feeding 13 pigeons with polished rice and water, which are in agreement with those of previous observers. They then prepared a watery extract of the spinal cord of the ox so that one cc. of this corresponded to four grams of dry cord. 15 pigeons were fed upon polished rice and were treated with this extract. 12 pigeons were also treated with yeast and nucleic acid. As a result of these experiments they came to the following conclusions.—That a long course of observations on fowls suffering from polyneuritis is necessary if positive conclusions are to be reached as to the curative effect of any measure, and many birds must be experimented with. The administration of yeast, nucleic acid and thymine have no effect on the course of experimental beriberi, showing that the anti-neuritic base of FUNK is hardly a derivative of nucleic acid. The aqueous extract of autolysed spinal cord contains an anti-neuritic substance which cures symptoms of polyneuritis in rice-fed birds in daily doses equal to four grams of dried cord, but it cannot restore metabolism nor enable the affected birds to recover their body weight, which bears out the finding of COOPER when using extracts of other foodstuffs. The anti-neuritic substance is present in greater amounts in the nerve fibres than in the nerve cells and is produced by autolysis, for in the fresh cord relatively small amounts only are present.

[Yeast and nucleic acid have been shown by many observers to have a distinct effect on the birds under experiment; therefore the statement in the conclusions given by the authors requires some explanation. COOPER found that half gram doses of dried yeast daily were quite enough to prevent polyneuritis and also to maintain the body weight of birds fed on polished rice (this *Bulletin*, Vol. 1. p. 481).]

P. W. B.-S.

WELLMAN (Creighton), EUSTIS (A. C.), & SCOTT (L. C.). **The Rapid Cure of Polyneuritis Gallinarum by Intramuscular Injection of a Substance isolated from Rice.—Note on the Pathology of the Disease. A Preliminary Report.**—*Amer. Jl. Trop. Diseases & Prev. Med.* 1913. Oct. Vol. 1. No. 4. pp. 295-299.

This is a preliminary report of investigations which are being carried out in the Tulane University laboratories—some of which have previously been reported. Extracts of rice polishings were prepared on the lines laid down by FUNK and these were given intramuscularly to fowls, mostly far advanced in the disease. The number of cures directly traceable to the injections in the first series was small (2 out of 7) but these are stated to have been very convincing. The results of the pathological examination of

the fowls which died of the disease agree generally with those recorded by VEDDER & CLARK, but it is pointed out that possibly the cause of the convulsions, from which the fowls suffered, may be spinal irritation brought about by subdural haematomas which were generally present along the cord, together with extravasations of blood over the cerebral lobes.

P. W. B.-S.

YAMAGIWA (K.), KOYANO (T.), MIDORIGAWA (H.), & MOGI (T.).  
**Experimental Study on the Cause and Nature of Beriberi. Report II.**—*Sei-I-Kwai Med. Jl.* 1913. Aug. 10. Vol. 32. No. 8. p. 110. [The original in No. 12, Vol. 27, 1913, of *Jl. Tokyo Med. Assoc.*]

The authors made experiments on fowls in order to decide whether substances in bran are capable of preventing acid fermentation of rice, and also whether the poison produced by fermentation of rice can be neutralised or destroyed by substances in the bran.

They came to the following conclusions:—

"1. The bran and its ingredients are capable of preventing the acid fermentation of the polished rice.

"2. The bran and its ingredients appear to be incapable of neutralising or destroying the already formed poison of the fermented solution of rice.

"3. There is no difference in time in the recovery of the fowls, which are suffering from the beriberi-like disease caused by the feeding with the polished rice, by treating them with unpolished rice or with unpolished rice + Oryzanin injection. (Oryzanin is a substance isolated from the bran by SHIMOMURA, ODAKE, & SUZUKI as its active principle.)"

P. W. B.-S.

## PAPPATACI FEVER.

BELLILE (P.). *Etude sur la Fièvre des Phlébotomes.*—*Arch. de Méd. et Pharm. Navales.* 1913. July. Vol. 100. No. 7. pp. 5-39. With 3 text-figs.

This is an excellent article on sandfly fever, in which is condensed all the most important information relating to the subject; in it also is included the author's own experience of the disease among the personnel of the French Man-of-War, "Amiral-Charner," while it was stationed at Crete during the years 1910-1912. The ships of France, Italy, Russia and England were sending by turn a detachment of marines to guard the international colours on Suda Island. More than half of the men who performed this duty during the summer were attacked with sandfly fever. The conditions were as precise as a laboratory experiment. No one who remained on board the "Amiral-Charner" was seized although there were mosquitoes in abundance in the ship, but there were no sandflies. On Suda Island on the other hand sandflies were numerous but mosquitoes were few. Thirty eight temperature charts are given in which it is seen that the fever lasts usually two or three days, but in one the temperature was not normal until the eighth day. The highest point attained seldom reached 104° F. Nineteen charts of cases which occurred among the crew of the "Conde" are inserted. They are those of the sandfly infection, although at the time the ailment was recorded as ague. The author quotes Fleet-Surgeon KILROY and Surgeon ADSHEAD's experimental work which was carried out at Suda Island, and reproduces the charts which appeared in their paper (see this *Bulletin*, Vol. 2. p. 25). On many occasions he has made cultures and microscopical examinations of the blood, but only with negative results. He notes that Phlebotomus fever differs from dengue in the absence of rashes and of the secondary rise of temperature. The outbreaks of dengue are more sudden and wide-spread. In 1902 a man-of-war stationed at Cochin-China was rendered ineffective by an epidemic of dengue which swept through it. Those who are not conversant with the literature of Phlebotomus fever often confound this infection with ague; the disease passes under this name among the inhabitants of Crete.

After the doors and windows of the guard room on Suda Island had been protected with fine-mesh wire gauze, and the men slept under muslin nets, the incidence of sandfly fever was lessened. Before these measures were taken 14 out of 19 men had been attacked; afterwards 5 cases only occurred among 42 marines.

C. Birt.

**Annual Report of the Sanitary Commissioner with the Government of India for 1911.**—1913. Calcutta: Superintendent Government Printing, India. [Sandfly fever. pp. 10, 18, 19, and App. pp. xii, xxiv.]

**Annual Return of the European Army of India, of the Indian Army and of the Jail Population for the Year 1911.**—[Sandfly fever. pp. 9-16, 19-21, 24, 29-39, 41-43, 52-61, 72.]

In the returns of the European officers, men, women and children stationed in India, for the year 1911, 1,457 attacks of sandfly fever



are registered. Among the Indian troops 114 cases only are recorded, but we find that there were 832 admissions for "pyrexia of uncertain origin" from Indian troops quartered in stations where sandfly fever was prevalent in the British regiments. Since 600 of these attacks occurred in the sandfly season, it is probable that they would have been rightly designated sandfly fever in the majority of instances. Sandfly fever was epidemic in the British barracks at Lucknow, but there were no admissions of Indian soldiers recorded under this heading, yet 77 men of the 74th Punjabis were attacked during the sandfly season with a fever lasting three days. At Kila Drosh and Chitral there are annual epidemics of sandfly fever, but 194 cases of febricula which occurred in these stations in 1911 were returned under the heading of "pyrexia of uncertain origin," no sandfly fever being shown. It is clear therefore, that in order to obtain the true incidence of sandfly fever in India we must scrutinize the tables of the seasonal prevalence of "pyrexia of uncertain origin," remembering that sandfly fever prevails during the hot and dry months.

In 1911 the number of admissions for "pyrexia of uncertain origin" among the European troops was 1914; among the Indian 4,066. 1,313 attacks in the British and 2,551 of the Indian occurred during the period April to September. It is probable that sandfly fever was the cause of the majority of these admissions during the hot weather. In the British army there was an outbreak of 133 cases of "pyrexia of uncertain origin" at Rangoon mostly during the dry months, January to May. At Bhamo 53 of our soldiers were attacked in June to September. 250 cases are reported from Lucknow, all except 39 in March to August; the maximum prevalence being in July in which the rainfall was 1.33in. only. Ambala had 92 cases; Ferozepore 26, 20 of which occurred in the hottest months; Lahore 101 admissions, 84 in May to October; Pashawar 161, all except 31 during the hot and dry months, March to August. Multan 55, the epidemic being almost limited to May to July; Secunderabad 43, 23 in May to July; Belgaum 24, in April to July; Poona 67, of which 46 occurred in April to August; Cherat 51, 39 in May to October; Aden 36, 26 in April to August.

In the Indian troops there were epidemics of "pyrexia of uncertain origin" at Mandalay 254 cases, 186 in June to August; in Bhamo 129, all except 33 in July to September; Lucknow 274, 226 in June to November; Lahore 142, 109 in July to November; Rawal Pindi 124; Nowshera 21; Pashawar 31 in May to August; Kohat 49, 38 in June to September; Edwardsbad 56, all but 8 in June and July; Dera Ismail Khan 65 in May to October; Jandola 108 in May to October; Secunderabad 123; Poona 133; Kirkee 130; Malakand 71 June to August; Quetta 147, mostly in May to October; Jask 56 in June to October; Aden 41.

Six thousand would be an approximate estimate of the number of attacks of sandfly fever which occurred in the European and Indian troops during the year 1911. Outbreaks of "pyrexia of uncertain origin" which arose in the rainy season have been excluded from this estimate; such were reported in Calcutta, Bombay and Rangoon; they were probably mosquito-borne, although only 69 cases

of dengue were recorded among the British regiments during the year.

In the British army alone there was a loss of 32,000 days' service on account of "pyrexia of uncertain origin."

[It is probable that in the great majority of cases registered "pyrexia of uncertain origin" the fever arises from a virus conveyed by the mosquito or sandfly, or from infections by the enteric group of micro-organisms. Until the cause of the outbreak has been defined, aims at prevention may be misdirected; it is therefore of economic importance to ascertain the origin in every instance.]

C. B.

i. LOUGHINAN (W. F. M.). *Phlebotomus* in Aden.—*Jl. Roy. Army Med. Corps*. 1913. July. Vol. 21. No. 1. p. 92.

ii. *Phlebotomus* Fever and Papataci Flies in Aden.—*Ibid.* Oct. No. 4. pp. 402-405.

i. A *Phlebotomus* has been found in Aden, and three cases of sandfly fever were observed between February and May, 1913.

ii. Before it was known that certain short febrile illnesses occurring in the tropics were caused by the sandfly, it was recognised that these cases were distinct from ague, and they were designated "simple continued fever" or "pyrexia of uncertain origin." Even at the present time many attacks of sandfly fever are still included under such headings.

During the years 1907-1912 inclusive there have been recorded among the British troops stationed at Aden 600 admissions for "simple continued fever," or "pyrexia of uncertain origin"; 713 for ague; and 11 only for enteric fever. Seventy eight per cent. of the 600 cases in the first class occurred in the months of May to October when sandflies are most numerous; 40, 167, 135, 74, 63, and 76 are the figures for the months May to October respectively. In two thirds of 476 of these attacks, concerning which information is available, the duration of the pyrexia was 1 to 4 days; in only 25 of the remainder was the temperature raised for 8 to 10 days. In 1912 the 18th Indian Infantry stationed at the Crater, Aden, suffered severely from sandfly fever. June and July were the months of greatest incidence, when 95 out of the total of 108 cases were admitted to hospital. Malarial parasites were absent from the blood of all; only 7 admissions for ague are recorded during the year in this regiment.

The symptoms of sandfly fever at Aden vary considerably as elsewhere. Vomiting and profuse diarrhoea are not infrequent; hyperaemia of the fauces, slow pulse, leucopenia with relative increase in the large mononuclears, and a fall of the temperature by lysis identify this fever with the sandfly infection in other parts of the world.

The Aden sandfly is the *Phlebotomus minutus*, and is present throughout the year, though most abundantly in the months of May to October. It has been captured in inhabited barracks, bungalows and native quarters, and in caves which are the abodes of camel men and sweepers.

Malaria at Aden is usually imported; no anophelines have been found there recently. Other mosquitoes are common. [It is probable that many of the febrile attacks which last five days or more are mosquito-borne, since Aden is nearly exempt from enteric infections.]

C. B.

BIRT (C.). *Phlebotomus* Fever and Dengue.—*Trans. Soc. Trop. Med. & Hyg.* 1913. June. Vol. 6. No. 7. pp. 243-256.

The first part of this interesting paper is devoted to the history and etiology of *Phlebotomus* fever, special attention being drawn to its world wide distribution, and its great prevalence among naval and military forces. In the second part of the paper the author discusses the etiology of dengue with relation to sandfly fever, yellow fever, and ROGERS' "Seven day fever"; such terms as three day, seven day, and ten day fever are considered very unsatisfactory and often inaccurate, as no fever keeps time so precisely. There are considerable differences in the infectivity of the virus of sandfly fever and dengue. The *Phlebotomus* is not capable of transmitting sandfly fever virus until six days after feeding on a patient who is in the first day of his illness, whereas dengue has been conveyed by mosquitoes immediately after their meal of dengue blood, but the virus may survive in them for 8-27 days; they must however have taken the blood from the dengue patient from the second to the fifth day of the disease. It is suggested that it would be much better if infections were designated by their transmitting agency, as rat-flea fever, louse fevers etc.—which would bring vividly to the notice of the public these dangerous foes.

The following conclusions are given:—

"Dengue, phlebotomus and yellow fevers are caused by distinct but closely related kinds of virus. A fever lasting several days in which the examination of the blood for parasites, by culture, and by serum tests, is negative; characterised by slow pulse, leucopenia, and relative polynuclear decrease; occurring in a locality where mosquitoes are numerous, should be attributed to a virus carried by these flies, although some of the symptoms significant of dengue or yellow fever may be wanting."

[It must be remembered that bugs, fleas etc. may carry more than one disease; hence this method of nomenclature would cause confusion.]

P. W. Bassett-Smith.

#### PHLEBOTOMUS AND ITS BIONOMICS.

KING (Harold H.). On the Bionomics of the Sandflies (*Phlebotomus*) of Tokar, Anglo-Egyptian Sudan.—*Bull. Entomol. Research.* 1913. May. Vol. 4. Pt. 1. pp. 83-84.

Tokar is situated eighteen miles from Trinkitat on the Red Sea, and 58 miles south east of Suakin. More than 30,000 acres of alluvial soil, which are under water in July and August when the river is in flood, are sown with cotton. On drying, the earth cracks causing fissures several feet deep. This cultivated area is notorious for the plague of sandflies. The author visited the place

in October, 1912, and examined a plot under cotton nearly 2 miles from the nearest wall or building. The sandflies existed in myriads. As many as 15 would escape on turning over a clod of earth. A *Phlebotomus* larva was found at a depth of about 4 inches in damp earth near a crack.

A wagtail (*Motacilla alba*) feeds on sandflies at dawn before the insects have returned to shelter for the day.

C. B.

HOWLETT (F. M.). i. *The Breeding Places of Phlebotomus*.—*Proceedings of the Third Meeting of the General Malarial Committee held at Madras, Nov. 18, 19, & 20, 1912.* pp. 209-210. (1913. Simla: Government Central Branch Press.)

ii. *The Natural Host of Phlebotomus minutus*.—*Indian Jl. Med. Research.* 1913. July. Vol. 1. No. 1. pp. 34-38. With a map and 1 plate.

i. The author is Imperial Pathological Entomologist, and his observations were made at Pusa, Bengal.

The larvae of *Phlebotomus* are killed by a comparatively short exposure to dry air; they are never found in dry or nearly dry earth. They require a moderate degree of moisture—such as that which generally occurs in English garden mould—protection from light, and the presence of nitrogenous refuse, the debris of dead insects, decayed fungi, and possibly insect and other dejecta; but an excess of this material is harmful. Bricks, stones, tiles, and cement form their common breeding haunts: but they have not been found in the brick work of wells, nor in latrines in Pusa.

Phlebotomi are often seen on lizards which do not resent their bites; it seems not improbable that they breed in the crannies in which lizards hide, and that the excreta of the latter may afford nourishment for the larvae. Howlett has seen phlebotomi in the surface galleries of termites' nests, and FLETCHER has made similar observations in Madras.

The number of larvae and pupae discovered at Pusa is small. A larva was found in the nearly dried mud of a cement channel leading from a well reservoir; another was captured in a small heap of kitchen refuse near a wall. Four larvae and seven empty pupa cases were collected from the damp earth between the bricks forming a platform for a "tulsi" plant in the courtyard of a house. The bricks were partially covered with algal growth, and in the crevices between them were ants, their larvae and nymphs, woodlice, larvae of Mycetophilidae, mites and nematode worms, with insect remains of many kinds on which the larvae were feeding. Five larvae, 3 pupae, and 23 empty cases were found in a small heap of bricks, tiles and other rubbish on waste land 30 yards from houses. The pupae were mostly on the edges of the tiles and bricks. Six adult flies emerged from damp algal muddy material collected from the sides of an open reservoir where water from several gutters leading from houses accumulates. The period of the year when larvae were caught was May to October.

ii. The distribution of the gecko or wall-lizard and of *P. minutus*, and their respective habitats, are closely similar. The *P. minutus*

breeds at Pusa at all times of the year, but it attacks man in the summer only. Since a repast of blood appears to be essential for the fertilization of the eggs, the phlebotomus must obtain this from some non-human source during the winter. The insect frequently may be seen sucking the blood of the common Indian lizards, *Hemidactylus gleadowii* and *Hemidactylus coctaei*, which do not appear to heed its attacks. In laboratory experiments it is easier to induce *P. minutus* to bite a lizard than a man; moreover the most suitable food for the larvae is lizards' faeces, or decayed mushrooms mixed with damp earth.

On examining the blood contained in the stomachs of 40 *P. minutus* captured in living apartments at the end of April and the beginning of May, lizards' red blood corpuscles were found in 24, and human in 11.

On two occasions a sand-fly was observed on the common toad, *Bufo melanosticticus*, but in 6 or 8 experiments the *P. minutus* sucked the blood of toads once only. Geckos therefore should be regarded as the natural hosts of *P. minutus*.

[ROUBAUD also noticed many *P. minutus* on a lizard; see this *Bulletin*, Vol. 2. p. 28.]

C. B.

WENYON (C. M.). The Length of Life of Phlebotomus in Captivity. A Note on a Method of Keeping the Flies alive for Experimental Work.—*Jl. London School of Trop. Med.* 1913. Nov. Vol. 2. Part 3. pp. 170-171.

At Malta during the summer of this year porous earthenware vessels shaped like flower pots, standing in water and covered with the finest muslin, were used to confine sandflies. They were set free every second, third, or fourth day in a net of dimensions 7 x 7 x 7 ft. They almost invariably settled on the cotton top, and were re-captured by placing 2 x 3/4 in. test tubes over them. They usually fed when the tube was inverted over the skin.

In the most successful experiment eight male and seven female sandflies were enclosed in the earthenware pot; all the males died within four days. The females were fed every other day. One died after nineteen days captivity; three survived 28 to 46 days. None of the flies deposited eggs, although on dissection all contained mature ova.

The results of the other experiments are not given.

C. B.

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## DENGUE AND UNCLASSED FEVERS.

ROUCHÉ. Note sur une Epidémie de Dengue à bord de la "Manche" en 1911.—*Arch. de Méd. et Pharm. Navales.* 1913. June. Vol. 99. No. 6. pp. 450-461.

In 1906, 1907 and 1908 epidemics of dengue were experienced on board the transport "Manche" while in Tonkinese waters, a very large proportion of the Europeans contracting the disease in each

epidemic. A further epidemic broke out in 1911, differing slightly from the former ones and in the early cases the diagnosis from malaria and influenza was difficult. The ship left Saigon on the 17th August, the first case was noted on the 6th of September and the last on the 3rd of November at Haiphong. 91 out of a total of 160 were affected, none of the natives on board contracted the disease either in this or the early epidemics and the incidence among the officers was very high (10 out of 12); the two who did not contract the disease in 1911 had already suffered from dengue in 1907, so that undoubtedly one attack gives definite protection. During the time of the epidemic mosquitoes (*C. fatigans*) were very abundant on board the ship and they evidently played an important rôle in the etiology, though from whence the infection was derived was not ascertained, neither were any bacilli or parasites found in the blood of the patients. The onset of the disease was abrupt; the fever lasted about 5-6 days; four types of temperature curves are described, the typical double fever was present in about a third of the cases and terminal rashes were rare. The convalescence was sometimes prolonged but there was no mortality. The prophylactic measures indicated were destruction of the mosquitoes and isolation of the infected.

P. W. Bassett-Smith.

**KHAN (Soleiman).** **Dengue at Meerut.** [Correspondence.] — *Indian Med. Gaz.* 1913. May. Vol. 48. No. 5. p. 204.

In the recent epidemic of dengue, which like that of 1872 spread from east to north west of India, Meerut suffered severely. From August to the middle of September nine-tenths of the population contracted the disease; the writer himself treated 1,400 cases as out-patients. The symptoms were very definite, the onset was sudden, the rash generally appeared on the second day, and the crisis occurred about the fourth. The incubative period was not more than five days, there was no mortality, but relapses were fairly common. It was apparently not spread by food or water, though very infectious; it is noted that there were few mosquitoes but swarms of flies at the time. No experimental work was carried out relating to the etiology of the disease.

P. W. B.-S.

**Dengue or Seven-Days Fever.** Discussion at the Asiatic [Society of Bengal]. — *Indian Med. Gaz.* 1913. May. Vol. 48. No. 5. pp. 199-202.

In a discussion on HOSSACK'S paper on Dengue read before the Asiatic Society of Bengal (see this *Bulletin*, Vol. 2. p. 30) many speakers agreed that there was no etiological difference between dengue and seven day fever. ROGERS strongly opposed this view, chiefly on epidemiological grounds, and maintains that three day, seven day fever, and epidemic dengue are distinct diseases.

P. W. B.-S.

LALOR (N. P. O'Gorman). **A Paper on the Aetiological Relationships of Seven-Day Fever. A Suggestion.**—*Proceedings of the Third Meeting of the General Malarial Committee held at Madras, Nov. 18, 19, & 20, 1912.* pp. 207-208. (1913. Simla: Govt. Central Branch Press.)

In the course of a *Stegomyia* survey of Rangoon the author noticed that seven day fever appeared to be localised in its distribution in a manner which suggested its association with the local prevalence of *Stegomyia fasciata*. He points out the similarity of the characters of this fever with yellow fever, both being probably due to filter passing mosquito carried viruses, and he suggests that the prevalence of this seven day fever in seaport towns might act as a weather gauge in India and Burma for estimating the risk of dissemination of imported yellow fever, a risk which will be more definite after the opening of the Panama Canal. He also suggests a number of aetiological experiments on the lines of those carried out with yellow fever to prove if this *Stegomyia* is really the carrier of the short local fever—which he considers to be a different entity from dengue.

P. W. B.-S.

ELLIOTT (M. S.). **A Case of Six-Day Fever.**—*U.S. Naval Med. Bull.* 1913. July. Vol. 7. No. 3. pp. 412-413.

The clinical course of the case coincides closely with seven day fever as described by CASTELLANI, and in most points with the cases of DEEKS from the Panama zone. The patient visited both Panama and Colon. The incubative period was 6-9 days, the temperature curve was characteristic, the pulse rate was slow and there was a marked leucopenia with a reduction of the polynuclear leucocytes, but the spleen was not enlarged and no eruption was noted. It was the only case which developed in the ship and the patient did not remember being bitten by any insects.

P. W. B.-S.

GABBI (U.). **Sulle "Febbri non Classificate" a Tripoli.**—*Commissione Governativa per lo Studio delle Malattie Tropicali nella Libia. Malattie infettive e Malattie cutanee. 2° Contributo ad Opera di U. GABBI, F. SCORDO, G. RIZZUTI.* (Ministero dell' Interno e d. Guerra. Direz. Gen. d. Sanità Pubblica.) pp. 23-30. 1913. Messina: Stab. Tipografico Guerriera.

The author gives a review of the literature of sub-tropical and tropical undetermined fevers. He then points out how difficult the clinical diagnosis of these fevers of "obscure origin" is from irregular typhoid, para-typhoid, colon infections and undulant fever, from all of which they may be separated by serum diagnostic reactions. Such forms were common in Tripoli and he describes two cases in Bedouin arabs, with irregular fever, slight gastro-intestinal symptoms, markedly enlarged spleens and negative serum reactions to all common organisms; both made good

recoveries without any particular treatment. There was no doubt that the patients were suffering from some infectious fever, probably a gastro-intestinal toxæmia. The presence of such infections is of great importance to all interested in civil and military medicine and sanitation; probably the dust laden winds in Tripoli are an important means of the distribution of the infecting agent, though food, cutaneous inoculation, and the intermediate action of flies must be considered. A very good bibliography is given.

P. W. B.-S.

### VERRUGA PERUVIANA.

STRONG (R. P.), TYZZER (E. E.), BRUES (C. T.), SELLARDS (A. W.), & GASTIABURU (J. C.). *Verruga Peruviana, Oroya Fever, and Uta. Preliminary Report of the First Expedition to South America from the Department of Tropical Medicine of Harvard University.*—*Jl. Amer. Med. Assoc.* 1913. Nov. 8. Vol. 61. No. 19. pp. 1713-1716.

The authors give an historical account of verruga and Oroya or Carrion's fever, records of which can be traced as far back as the 17th century. Up to the present time it has been generally accepted that the two conditions are stages of a single disease, the acute fever being followed by the eruption. From their investigations they have come to the conclusion that verruga and Oroya fever are two distinct diseases, though not infrequently occurring in the same person.

Oroya fever is often very fatal and always associated with marked blood changes, especially in the number of nucleated red cells present and in the appearance in the protoplasm of the stained red cells of peculiar rod-shape inclusions called "Barton's bodies." Similar rods with some spherical and pear-shaped bodies were observed in this investigation in the fresh cells, when they appeared to possess definite motility, and were often very abundant in single cells. In stained specimens the ends of the rod-shaped forms were intensely coloured, having a chromatin appearance, and beading was seen. The spherical forms were from 0.3 to 1.0 micron in diameter and sometimes showed chromatin granules. The red cells may contain as many as 30 of these elements, which may also be seen in the various nucleated forms. It is presumed that these bodies destroy the red cells and produce the marked anaemia. The bodies appear to be closely allied to those described by GRAHAM SMITH in the blood of moles and classed by BRUMPT as a parasite under the name of *Grahamella*. The authors propose to create a new genus "*Bartonia*" and name the parasite of Oroya fever *Bartonia bacilliformis*. Detailed characters of the genus and species are given.

*Verruga peruviana* is a distinct specific disease differing from frambesia and syphilis, inoculable from man to man, and man to animals (monkeys, dogs and rabbits). A crucial experiment was



carried out on a Chilean volunteer. He was thoroughly inoculated through the scarified skin with the nodules of two verruga patients; sixteen days after local papules appeared and increased until the 35th day, when they were removed. No generalised eruption, no fever and no changes in the blood cells as in Oroya fever occurred.

[From the above it will be seen that the authors have thrown over the paratyphoid theory for Oroya fever, and emphatically accept as the cause of the disease a protozoal parasite, originally described by BARTON, GASTIABURU and others, but which NICOLLE and others looked upon as a product of cell degeneration. Further work on the subject will be required to establish this new genus and the term *Bartonia* will not stand as it is already in use as a generic name.]

P. W. Bassett-Smith.

DARLING (S. T.). *Verruca Peruana*. — *Jl. Amer. Med. Assoc.* 1911. Dec. 23. Vol. 57. pp. 2071-2074.

In this paper (published in 1911) Darling gives fully the historical, clinical and etiological factors of verruga. According to Darling there are two types (1) A malignant form known as Oroya fever or Carrion's disease, general, without eruptive stage; (2) A benign form, with slight mortality and frequent eruption. In the section dealing with the etiology, the X bodies described by BARTON in 1905 and thought by him to be parasites are fully described, and their relationship to *Babesia* and the endo-corpuscular bodies of GRAHAM SMITH (found in moles) pointed out. The very extraordinary alteration in the blood cells and the abundance of the nucleated forms found in the severe type of the fever are described. The knowledge of the limitation of the infective area and the common belief that infection occurred at night led BARTON to insist that the railway workers should sleep in houses outside this area; after this was done there was a complete cessation of cases.

Darling considered that the endo-corpuscular bodies may be the cause of the disease.

P. W. B.-S.

TOWNSEND (C. H. T.). i. *La Titira es Transmisora de la Verruga*. [Transmission of Verruga by the 'Titira.']—*Cronica Med.* [Lima.] 1913. June 30. Vol. 30. No. 588. pp. 210-211.

ii. *Progress in the Study of Verruga Transmission by Bloodsuckers*. — *Bull. Entomol. Research.* 1913. Sept. Vol. 4. Part 2. pp. 125-128. With 3 plates

iii. *The Transmission of Verruga by Phlebotomus*.—*Jl. Amer. Med. Assoc.* 1913. Nov. 8. Vol. 61. No. 19. pp 1717-1718.

i. In an investigation which the author as Government Entomologist of Peru carried out, with the object of solving the problem of the means of transmission of verruga, the results of experiments with ticks were completely negative and he also excludes *Tabanus*, *Stomoxys*, fleas, lice, bugs, and leeches. He made a personal inspection of the blood-sucking fauna of the infected district of San Bartolome, finding, besides mosquitoes, that a species of *Phlebotomus* was very common. On epidemiological grounds he believes that this fly is the transmitter of

the disease, for it was found to be most abundant in those hot steamy ravines with rank vegetation, where verruga was most prevalent; he considers that these ravines are ideal places for the flies to breed in. No experimental proof has so far been obtained. It is stated that for years past the natives of Peru have suspected a relationship between verruga and the Phlebotomus.

ii. The author describes the results of his observations to determine whether verruga is transmitted by bloodsuckers. No results were obtained to incriminate Phlebotomus, as sufficient numbers were not found owing to the season for the fly being nearly over; experiments are however to be carried out at the earliest possible opportunity. The plates show views of the Verrugas canyon, the Verrugas bridge, the building of which cost thousands of lives, and the canyons at Chosica below the verruga zone, where there is an absence of cover for Phlebotomus.

iii. To prove the Phlebotomus theory of transmission he obtained two hairless Mexican dogs. After they had been kept under observation for nearly three months, one was inoculated subcutaneously with the ground up bodies of twenty female Phlebotomus collected the night before in the Verruga canyon. On the 5th day the injected dog had lost flesh and appeared ill. The blood showed endo-globular bodies like BARTON'S X-bodies, nucleated red cells and broken down red cells. On the 6th day a typical nodular eruption was noticed on the right hind foot. On the 8th day the dog began to improve. Smears from one of the papules showed bodies resembling Leishmania. The control dog remained in perfect health. This being the first instance of experimental transmission of the disease by means of insects it is, the author writes, of great interest; the very short incubative period, absence of fever and early eruption are remarkable. The author states that dogs are less susceptible than man in that the disease runs a more benign course; mild cases in man being associated with little fever and early appearance of the eruption.

[If this experiment is confirmed by other investigators it goes far to prove that the fever, with its peculiar blood characters, and verruga are only stages of the same disease, but it is in direct opposition to the views of STRONG and others expressed above.]

P. W. B.-S.

DA ROCHA-LIMA (H.). Zur Histologie der Verruga peruviana.—*Verhandlungen der Deut. Pathologischen Gesellschaft.* 1913. April. pp. 409-416. With 1 plate and 2 text-figs.

The author gives a short history of the disease known as verruga or Oroya fever and draws attention to the occurrence of a rather similar condition in Brazil called Bassewitz's disease; the latter however does not show clinically the acute febrile onset common in Peruvian cases. At Hamburg, the author and others were able carefully to observe a recent case of verruga, and a full description of the histological characters of the growths which were removed

is given. He states that he was only able to examine the skin nodules, and that his views as to their structure differ to some extent from those of most authorities. His conclusions at the end of the paper are to this effect:—A verruga nodule consists of endothelial offshoots, blood vessels, and a more or less oedematous or haemorrhagic and lymphatic (eventually leucocytic) infiltrated connective tissue, whose relative amounts and distribution in the nodes or in different parts of the same node, may show great variation, so that at one time a granulating, at another a sarcomatous, myxomatous or angiomatous appearance may be presented; of known affections the telangiectatic granulomata described by KONJETZNY\* are those possessing the closest resemblance to the verruga tumours. Contagious circumscribed angiofibroma of the skin, found in south Brazil, and conditions of human botryomycosis though histogenetically related processes are not quite identical, differing principally by the fact that in telangiomatous granulomata the construction of vessels, in verruga the hyperplasia of the endothelium, appears to be the characteristic event.

P. W. B.-S.

\* KONJETZNY. *München. Med. Wochenschr.* 1912. Vol. 59. pp. 2219-2221.

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## HELMINTHIASIS.

AMERICAN SOCIETY OF TROPICAL MEDICINE. Is the Importance of Intestinal Parasites in Tropical Pathology Exaggerated? [Secretary's Abstract, by John M. SWAN, of the discussion at the 10th Annual Meeting, 1913.] — *Amer. Jl. Trop. Diseases & Preventive Med.* 1913. Aug. Vol. 1. No. 2. pp. 169-172.

In Dr. E. R. STITT's opinion the reduced death rate in Bilibid Prison, Philippine Is., from 75 to 9 per 1,000 after quarantining of persons harbouring intestinal parasites, indicates that some importance is to be attached to their presence.

Dr. C. WELLMAN some years ago investigated a disease among the fishermen in Western Africa which was characterised by a severe anaemia and was attended by a high mortality. The cause was found to be the *Dibothriocephalus latus*. In one district in West Africa 13 per cent. of the population was incapacitated by uncinariasis. The activities of the Rockefeller Commission have been highly beneficial in reducing hookworm disease. In highly malarial countries the presence of intestinal parasites affects the mortality in two ways. (a) Hookworm infection increases the mortality from malaria, and (b) over-zealous treatment of hookworm disease increases the mortality from malaria. Some parasites such as *S. subtilis* [*Trichostrongylus instabilis* = *T. colubriformis*], *H. nana*, *T. trichiura*, etc. have been taken too seriously. It is possible that heavy infections with these may produce some anaemia. In some districts in the Tropics 100 per cent. of the children suffer from *Ascaris* infection. This worm produces convulsions and even stimulates recrudescence of malaria. He believed that the importance of intestinal parasites is not exaggerated.

Dr. NICHOLS considered that the intestinal parasites have not the serious influence that other [protozoal] parasites have because they cannot reproduce themselves in the intestines. It is perhaps necessary to exaggerate the importance of these parasites as a text for agitation to interest the public and legislators in necessary public health measures, but it is not essential for medical men to accept this exaggeration.

Dr. CRAIG thought that the hookworm is the only intestinal worm of great importance in tropical pathology. The schistosome is important but its *locale* is limited and the number of cases is small. In the southern parts of the United States the importance of the hookworm is being exaggerated. The hookworm campaign has improved the health of the people in districts in which there is no malaria, but whether anything has been accomplished in districts in which malaria is present is doubtful.

Dr. E. STITT believed that the discovery of ova in the faeces frequently puts the clinician off the track. According to Dr. E. F. OTIS of Porto Rico it does appear that infection with intestinal parasites produces a physical, vital and nervous effect that classifies children, particularly, as deficient. Certain intestinal parasites appear to produce a toxæmia.

R. T. Leiper.

LEIPER (Robert T.). **Observations on Certain Helminths of Man.**—*Trans. Soc. Trop. Med. & Hyg.* 1913. July. Vol. 6. No. 8. pp. 265-297. With 36 text-figs.

The paper is based upon a demonstration given before the Society of Tropical Medicine and Hygiene on June 20th, 1913. New points of interest relating to some of the parasitic helminths are given and diagrams, where those help the subject, are included. Writing on the *Lagochilascaris minor*, Leiper thinks these worms were undoubted wandering forms and have not found the intestine of man a congenial habitat. Possibly they are normal intestinal parasites of the cat in the West Indies and this idea is borne out by a closely allied form, *L. major*, having been found in the East African lion by the author.

Two instances of the occurrence of *Ascaris lumbricoides* in the dog are given, these are regarded merely as rare cases of accidental infection. As regards the *Physaloptera mordens* it is suggested that the monkey is most probably the normal "reservoir host." A new pair of papillae is described in *Acanthocheilonema perstans* (= *Filaria perstans*). This is said to lie between the solitary pair described by Low, and the posterior extremity. The pre- or, as the author terms it, perianal group consists of four pairs as noted by Low. Details are also given of the papillae and spicules of *Filaria loa* and *F. bancrofti*, diagrams showing these very clearly (figure).

*Oesophagostoma apiostomum* is said to be a common parasite of monkeys and produces in them dysenteric symptoms when the infection is severe. The first recognition of this species in man was made by Leiper from material collected by Fox in Northern Nigeria.

RAILLIET and HENRY propose to place *Triodontophorus deminutus* in a new genus, namely *Ternidens*. The name *Syngamus kingi* is given to the pair of "forked" worms found by KING, of St. Lucia, in the sputum of a patient. Correspondence has shown that the case was a genuine one. As regards the *Gnathostoma siamense*, it seems as if this was just the same as *G. spinigerum*.

Several new discoveries, some of great importance, have been made amongst the trematodes and these are discussed. Of these the most important is the finding of a new genus (*Yokogawa*) by the author. This is a new fluke from Japan, discovered by YOKOGAWA, and widely spread amongst the inhabitants of Korea, Formosa and Japan. KATSURADA had already placed this parasite in the genus *Tocotrema* but Leiper brings forward evidence to show that the character of the yolk glands is different from that of that genus and so has proposed the new genus *Yokogawa*, the name of the species now standing as *Yokogawa yokogawa*. Two interesting cases of infection with *Heterophyes* from the far East are mentioned. The position of *Opisthorchis neverca* still gives rise to trouble. We have now arrived, according to Leiper, at the following state of affairs:—" *Opisthorchis neverca* " as it occurs in man is placed in the genus *Amphimerus*, Barker, and becomes *Amphimerus neverca* (Braun, 1903), Barker 1911. The

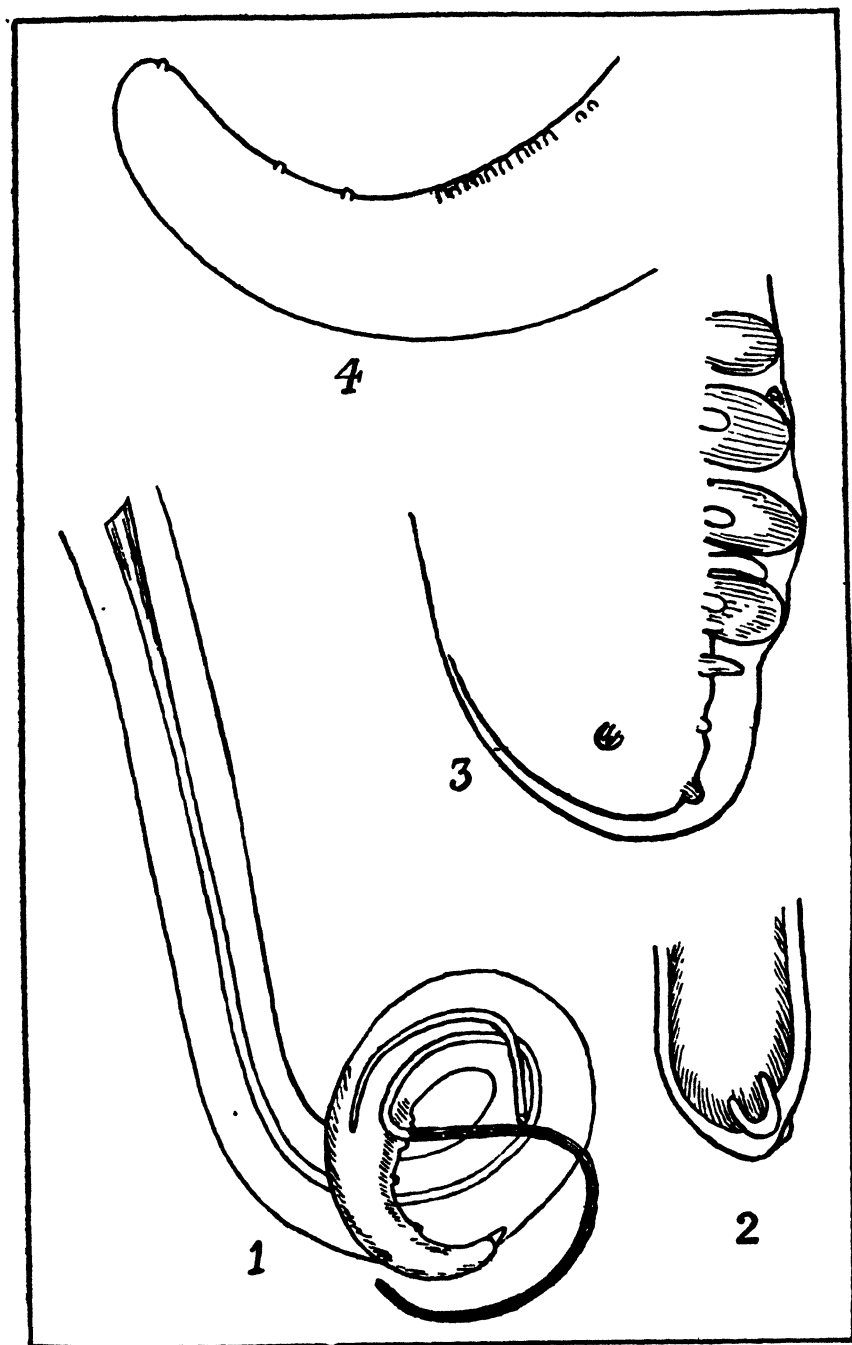


Fig. 1. Tail of *A. perstans* showing details of spicules and papillae.

Fig. 2. Posterior end of *F. oxzardi* (*denarquayi*) [type specimen] showing paired large fleshy papillae.

Fig. 3. Lateral view of tail of male *Loa loa* showing papillae, noted by LANE and the author.

Fig. 4. Posterior extremity of [male *F. bancrofti*] giving relative positions of the papillae.

"*Opisthorchis noverca*" of the dog is now *Paropisthorchis caninus* (Barker 1911), Stephens, 1912, with a stillborn synonym, *Paropisthorchis indicus*, Stephens, 1913." Leiper believes that these forms belong to one and the same species.

Regarding *Fasciolopsis* the following facts are fairly clear, viz. that the pig acts as a host of this genus and that spines are present on the skin in these forms. KWAN's fluke is not regarded as a distinct species. The only accepted characters distinguishing *F. buski* from other forms are (1) relative elongation, (2) lack of "muscularity," (3) absence of spines from the skin.

*Fascioletta ilocana* first described from the Philippines by GARRISON, has recently been re-examined by ODHNER and transferred by him to the genus *Echinostoma*, RUD. This has been done on account of the general topography and especially of the presence of a collar of spines around the anterior end of the body which is characteristic of the *Echinostomidae*.

MATHIS and LEGER, it is stated, have solved the mystery of the true host of *Gastrodiscoides hominis*. These authorities have found that no fewer than five per cent. of the pigs in Annam are infected by these worms. *Cladorchis watsoni* has been relegated to a new genus, viz. *Watsonius* by STILES. Leiper from sections of some of the original material sent from Northern Nigeria had already come to the same conclusion.

[The paper is a very interesting one and brings into reasonable compass much recent work on human helminthiasis. Students of helminthology should therefore consult the original.]

G. C. Low.

LEIPER (R. T.). *Seven Helminthological Notes.* — *Jl. London School of Trop. Med.* 1913. Nov. Vol. 2. Pt. 3. pp. 175-178. With 1 fig.

The presence of guinea worm in Nyasaland and of *Heterophyes heterophyes* in cases from the far East is recorded, also that of *Hymenolepis diminuta* in a child from Grenada, West Indies, and of *Paragonimus westermanii* in India.

The author states that a fluke described by COBBOLD from the Mongoose (*Viverra zibetha*) as *Distomum compactum* is a *Paragonimus* and may be the same species as in man; if so, the nomenclature will require revision, *Paragonimus westermanii* giving way to *Paragonimus compactus*.

Reference is also made to a statement made by STIRR that the two spicules in *Necator americanus* are constantly fused; the author has verified this and finds further that the spicules differ in several respects from those of *Ankylostoma duodenale*. They are provided with wide lateral expansions which are transversely striated, giving each spicule the appearance of a triangular file. These expansions gradually disappear, and the spicules when united show a delicate smooth ledge without striae, but slightly granular. This continues to the tip and forms the triangular piece. The rod-like portions of the spicules support this cuticular blade, one spicule being slightly curled to give support to

the proximal angle of the barb, the other continuing for some distance towards the extreme point.

G. C. L.

KAY (J. A.). **A New Ovum and its Miracidium.**—*Practitioner*. 1913. Oct. Vol. 91. No. 4. (No. 544.) pp. 580-582. With 1 plate.

LEIPER (R. T.). **A Comment on Two Recent Articles on Helminth Infections in Man.** [Memoranda.]—*Brit. Med. Jl.* 1913. Nov. 15. p. 1302.

In the article by Kay bodies are described which were believed to be the ova and miracidia of a new trematode.

From the figures and description, Leiper has not the slightest hesitation in affirming that the bodies do not bear any resemblance or relation to either ova or miracidia of a trematode and that they are certainly not even of helminthic origin. The figures and portions of the description might best be accounted for by the presence of Rotifers in the water used to dilute the urine.

He also refers to the recently described, supposed indigenous case of filariasis in Devonshire, published in the *British Medical Journal* (see this *Bulletin*, Vol. 2, p. 103). On an examination of the slides he found that the supposed filariae were minute filaments of cotton wool which had contaminated the freshly made blood films.

He expresses the hope that the above cases will not find their way into the text-books and quotes '*Filaria gigas*' as a familiar example of the manner in which such records may become stereotyped in spite of repeated repudiation by workers on helminthology.\*

G. C. L.

CROWELL (B. C.) & HAMMACK (R. W.). **Intestinal Parasites encountered in Five Hundred Autopsies, with Reports of Cases.**—*Philippine Jl. of Science*. Sec. B., Trop Med. 1913. June. Vol. 8. No. 3. pp. 157-174.

A number of reports have been published bearing upon the incidence of the various intestinal parasites found in the Philippine Islands. These have been wholly based upon clinical examinations of the faeces. In the present paper the percentage incidence of the parasites is based solely, and for the first time apparently, upon postmortem records and taken from the files of the Pathological Department of the Manila College of Medicine and Surgery. During the year 583 consecutive autopsies were made and of these 83 have not been utilized. Of the 500 cases the overwhelming majority were Filipinos, but Americans, Europeans, Chinese, Japanese and East Indians are also included.

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\* If the editors of medical journals were to submit such papers to experts before accepting them, we should be spared much useless lumber. A. G. B.



The statistics were not checked by routine examination of the faeces and may have been affected somewhat by the fact that the majority of the cases came from a hospital where routine treatment for helminths is in vogue.

*Incidence of intestinal parasites in 500 Autopsies.*

	No. of cases.	Per cent.
<i>Ascaris</i> ... ..	206	41.2
Hookworm ... ..	83	16.6
<i>Trichuris trichiura</i> ...	172	34.4
<i>Oxyuris</i> ... ..	5	1.0
<i>Taenia saginata</i> ... ..	1	0.2
<i>Clonorchis sinensis</i> ...	2	0.4
<i>Schistosoma</i> ... ..	1	0.2

The view of BRAUN and others that *Ascaris* leave the human host in the course of febrile diseases is not substantiated by the series of cases of malaria, typhoid, plague, lobar pneumonia, and bacillary dysentery, in which the infection with *Ascaris* ranges from 26.6 per cent. to 66.6 per cent. In one instance an *Ascaris* was found in the vermiform appendix and in two cases numerous worms had passed through the bile duct into the liver, in one case giving rise to numerous abscesses, in the other to marked inflammation of the bile ducts. Live *Ascarides* were found in the middle ear, in the bile ducts, pancreatic duct, stomach, oesophagus and larynx but in all these cases these wanderings were believed to have taken place after death.

Trichuriasis occurred in 33.9 per cent. of the males and 35.4 per cent. of the females, while 15.53 per cent. of the cases were less than fifteen years old and 40.1 per cent. over that age. In two cases only were whipworms found in appendices removed on account of disease.

No example of the adult *T. solium* was encountered. This appeared surprising in view of the widespread occurrence of *Cysticerci* in the hogs in the Philippine Islands. In only one of 2,200 autopsies a case of infection of man with *Cysticercus cellulosae* was noted. [The *cysticerci* in the hogs possibly were the larvæ of a dog tapeworm, e.g., *C. tenuicollis*.]

R. T. L.

JOHNSTON (J. E. L.). **A Note on Helminthiasis in Bassa Province, Northern Nigeria.**—*Lancet*. 1913. Sept. 27. pp. 926-927.

An examination of the stools of thirty persons taken at random in Ankpa, the chief European station in the Bassa Province of Northern Nigeria, gave the following percentages of helminthic infection.

Single infection in 12 ... ..	(40 per cent.)
Double     "     "     6 ... ..	(20     "     "     )
Triple       "     "     1 ... ..	( 3     "     "     )
No           "     "     11 ... ..	(36.6,     "     "     )

The ova found were

Ankylostome (or <i>Necator</i> ) in 14 ...	(46.6 per cent.)
<i>Ascaris lumbricoides</i> ... ..	(20     "     "     )
<i>Taenia solium</i> ... ..	(13.3     "     "     )
<i>Trichuris trichiura</i> ... ..	(10     "     "     )

Both *Ankylostoma* and *Necator* were recovered after anthelmintics.

In 26 of the cases the blood was examined and an eosinophilia was noticeable in almost every case, rising in one instance to 56 per cent. This was seen not only in the cases with ankylostome infection but with the others also. In some cases it was, in the opinion of the author, probably attributable to those skin eruptions termed generally "craw-craw."

Many cases showed great relative increase in the lymphocytes [? large mononuclears], possibly indicative of a preceding infection with malaria.

Haemoglobin was roughly estimated:—

1	had	40	per	cent.	haemoglobin.
3	"	50	"	"	"
1	"	55	"	"	"
13	"	60	"	"	"
1	"	65	"	"	"
5	"	70	"	"	"
1	"	80	"	"	"

In the case in which only 40 per cent haemoglobin was found there was a large abscess besides an ankylostome infection.

The author believes that the Hausas are much less affected with ankylostomes than the Bassa pagans, and attaches importance to the fact that they use deep cemented lined pits for defaecation whereas the pagan tribes have no special arrangement.

Epigastric pain was an uncommon symptom in the above series; the earliest symptoms being dyspnoea and dizziness, especially in a bright light, followed by pains in the legs, oedema, etc. Several of these were extremely like cases of beriberi and in some ova were not found till the second or even third examination. [The association of these symptoms with so slight an infection of ankylostomes seems very questionable. It is possible that the Cestode ova observed were those of *Taenia saginata*. It is unfortunate that the number of leucocytes counted is not stated nor the method employed in the haemoglobin estimation.]

R T. L.

BOURRET. *Recherches sur le Parasitisme Intestinal, la Dysenterie et la Maladie du Sommeil à Saint-Louis (Sénégal).*—*Ann. d' Hyg. et Méd. Colon.* 1913. April-May-June. Vol. 16. No. 2. pp. 283-307.

The stools of 52 natives of St. Louis were examined in order to ascertain the diffusion of intestinal parasites among the general populace. 40 proved negative. One contained segments of *Taenia saginata*, two showed eggs of *Ascaris lumbricoides*. In three *Oxyuris* eggs were present while in two cases the eggs apparently of *Necator americanus* were found.

Of three Europeans two were negative and one showed infection with *Trichocephalus* [*Trichuris*] *trichiurus*.

R. T. L.

## SCHISTOSOMIASIS.

HARRISON (W. S.). **The Prognosis in Bilharziasis.**—*Jl. R. Army Med. Corps.* 1913. Oct. Vol. 21. No. 4. pp. 385-388.

The cases of bilharziasis contracted during the South African War have afforded unique and reliable information regarding the duration of an infection and the degree of disability which results. From a study of 466 of these cases it is concluded that bilharziasis is a much more prolonged disease than has been usually supposed. The earliest period at which recovery may be hoped for is from five to seven years after the onset of symptoms while it may last without relief for at least thirteen years. The direct and indirect mortality from bilharziasis among Europeans removed from the endemic area probably does not exceed one per cent. Out of the 466 cases blood had been absent from the urine from two or more years in 141, but in 23 of these ova were still present. In 133 cases blood was found intermittently and in quite a number of cases signs of infection returned after having been in abeyance for over two years. The course of many of the cases strongly suggested the possibility of reinfection from the patient's own urine. In 164 of the cases blood and ova were present in the urine continuously. In 5 of these the disease had lasted for 13 years, in 16 for 12 years and in 57 for 11 years. There were 28 deaths; 7 were possibly attributable to bilharziasis. Of these two, regarded as open to doubt, were "carcinoma of the bladder" and "internal haemorrhage." Although 8 deaths were due to tubercle this is not regarded as indicative of an increased tendency resulting from the bilharzia infection as official returns show that in England 29 per cent. of all deaths of males between the ages of 25 and 45 are due to phthisis.

R. T. L.

EDGAR (W. H.). **Yangtze Fever.**—*Jl. State Med.* 1913. Sept. Vol. 21. No. 9. pp. 542-553.

The terms Yangtze fever, river fever, Hankow fever or urticarial fever have been applied in the past by those practising in the foreign communities in the valley of the Yangtze-kiang, in China, to any attack of fever of an undetermined nature. It is now considered that the name Yangtze fever may be confined to a well marked condition which typically is a remittent form of pyrexia lasting from three to six weeks. For the first few days the patient feels out of sorts, and especially towards evening there is a headache and achings in the loins and limbs which become daily more pronounced. The temperature rises to 103° F. or so at nights, falling to about normal in the morning. The subjective symptoms set in usually shortly after noon and the height of the fever is generally reached by 6 p.m. Rigors are rare.

The temperature is suggestive of malaria but no parasites are to be seen in the blood. There is however a marked eosinophilia of from 12 per cent. to 50 per cent. which with the daily temperature is extremely characteristic if not actually pathognomonic of the condition. The temperature continues to rise daily to 103° F.

for a week and then gradually falls day by day, reaching normal during the third or fourth week. About half the cases have a further characteristic, *viz.*, a marked tendency to localized oedema. This occurs early and often is the first symptom. These lesions appear as large annular patches, hard to the touch and often accompanied by marked itching, resembling in fact ordinary urticaria. In some cases the lesions are transient, in others they continue throughout the fever. Localised oedema may occur also in the lungs, producing signs of consolidation and accompanied by a dry cough, rarely cyanosis and sudden dyspnoea.

A painless and transitory diarrhoea probably due to localized patches of oedema of the great intestine is often present at the onset; more usually however the stools are large and hard. In some cases dysenteric symptoms predominate and the ova of *S. japonicum* can be found in the faeces.

In addition to these major symptoms *anorexia* and *mental irritability* are especially marked.

During the incidence of the fever the patient loses much weight and strength and the resulting debility may be serious to those who already suffer from malaria or dysentery. The mortality is however *nil* and with a change of climate normal health is regained in two or three months.

As the condition chiefly attacks young men and children and as the victims always give a history of having bathed in creeks or waded in marshy ground while shooting, it was supposed that the fever was due to some organism found in muddy water, which probably gained entrance through the skin. Dr. O. T. LOGAN first noted the incidence of *S. japonicum* ova in the stools of a child suffering from "urticarial fever" with dysenteric symptoms and since then much evidence has been forthcoming to substantiate the theory that this parasite is the causative agent of the disease. Surgeon J. VERDON, R.N., is quoted as writing, from Kiukiang, the opinion that the cases under his care were due to some form of food poisoning.

Treatment is symptomatic. Quinine is useless at first but exercises a beneficial action after the third week or so. Acetylsalicylic acid in 10 or 15 gr. doses as the temperature begins to rise each day is unsurpassed for the pain, aching and headache. The urticaria may require a soothing lotion but an occasional Izal bath is more effective. The diet should be liberal after the first few days and a change to the coast should be made, if possible, as soon as the evening temperature remains normal.

R. T. L.

#### TAENIASIS.

DÉVÉ (F.). *Echinococcose secondaire embolique périphérique.*—*Compt. Rend. Soc. Biol.* 1913. July 25. Vol. 75. No. 27. pp. 100-102. With 1 text-fig.

Eleven years ago the author demonstrated the formation of pulmonary embolic or metastatic echinococcus cysts after the

experimental injection of hydatid material into a *vein* in the ear of a rabbit. A new experiment is now recorded to show that the injection of scolices into the carotid artery results in the development of embolic peripheral cysts.

R. T. L.

#### ANKYLOSTOMIASIS.

LEIPER (Robert T.). The Apparent Identity of *Agchylostoma Ceylanicum* (Looss, 1911), and *Agchylostoma Braziliense* (Faria, 1910).—*Jl. Trop. Med. & Hyg.* 1913. Nov. 1. Vol. 16. No. 21. pp. 334-335. With 1 text-fig.

LANE records the occurrence of *Agchylostoma ceylanicum* as an occasional parasite of man and an almost constant parasite of cats and dogs in Bengal. Specimens of these were sent to Leiper at the London School of Tropical Medicine and the difference in the subdivision of the dorsal ray was noticed. In *A. duodenale* the elongated dorsal ray is bifurcated near its tip and the subdivisions resolve themselves into three small finger-like branches; in *A. ceylanicum* the primary subdivisions resolve themselves into two terminal branches (see figure)

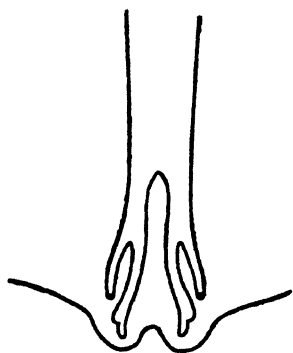


Fig. 1.

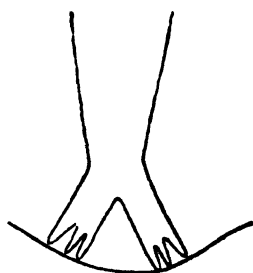


Fig. 2.

Fig. 1. Dorsal ray of *A. ceylanicum* vel *braziliense*. (Reproduced by permission from the *Jl. of Trop. Med. & Hyg.*)

Fig. 2. Dorsal ray of *A. duodenale*.

In a paper recently published by GOMES de FARIA in Brazil a new ankylostome was described. These parasites were found commonly in cats and in one instance in a dog. A comparison of LANE's figures, descriptions and material from Bengal with the paper and illustrations of FARIA from Brazil have led Leiper to conclude that these two species are identical. The common occurrence of the same parasite in cats and dogs in Bengal and Brazil suggests that the form will be met with in other parts of the world as well, but the author in examining his own material from cats and dogs from Africa has not so far encountered it. He suggests that workers in China should re-examine supposed

specimens of *A. duodenale* carefully in the light of the new facts now available.

A note is added at the end of the paper with regard to the spelling of the generic name *Agchylostoma*. The committee of the International Congress of Zoologists, now drawing up a list of generic names which shall be placed outside the possible operation of the Law of Priority, has adopted the spelling *Ancylostoma*. The author, however, points out that these International Rules apply solely to nomenclature and that corresponding terms such as Ankylostome and Ankylostomiasis do not come under their operation as no "Rules of Terminology" have yet been formulated. The spellings agchylostome, agchylostomiasis, ancylostome, and ancylostomiasis have no special authority, and may be discarded therefore as pedantic.

G. C. L.

MOORE (A.). Remarkable Emaciation in a Case of Hookworm Disease.—*Amer. Jl. of Trop. Diseases & Preventive Med.* 1913. Oct. Vol. 1. No. 4. pp. 294-295. With 2 illustrations.

Two photos illustrate the extraordinary emaciation which supervened in a professional trapper of 20 years of age apparently as a result of an ankylostome infection. A year previous the patient had had typhoid fever from which he never regained his strength. Since then treatment was followed under medical advice for tuberculosis and for pellagra. Distressing diarrhoea, complete anorexia and inability to retain nourishment were the outstanding clinical features of the case. These were promptly relieved by thymol. During four weeks spent in hospital there was rapid recovery.

R. T. L.

KEITH (R. D.). The Treatment of Ankylostomiasis.—*Lancet.* 1913. Oct. 18. pp. 1117-1118.

Watson's recent statement\* underestimates the importance of ankylostomiasis in Malaya, at least as regards Singapore and Johore. From a study of 70 cases the percentage of haemoglobin is considered a point of great importance. With the percentage in the neighbourhood of 60 the ova will usually disappear within a few days with suitable treatment. At 45 per cent. to 55 per cent. treatment will be more difficult and anywhere below 40 per cent. indicates that great patience will have to be exercised. When the haemoglobin drops to 10 per cent. to 20 per cent. weeks or months even may pass before recovery is effected.

The general condition may be improved with rest and diet, and the oedema may disappear although the haemoglobin index remains persistently at about its original level.

\* *Brit. Med. Jl.*, 1912. Nov. 2. p. 1201.

The ova do not disappear from the stools after treatment probably on account of their retention on the walls of the intestine, the survival of worms embedded in the mucus, and reinfections.

Beta-naphthol in 30 gr. doses the first thing every morning is thought from certain recent cases to be more effectual than thymol or eucalyptus. It is neither necessary nor even advantageous to starve the patient prior to the administration of the drug as there is not much food in the upper part of the intestine five or six hours after a meal.

R. T. L.

**YOSHIDA.** On the Haemolysis by Thymol.—*Sei-I-Kwai Med. Jl.* 1913. Oct. 10. Vol. 32. No. 10. (Whole No. 380.) pp. 125-126. (The Original in No. 3, Vol. 1, of the Japanese Med. Soc.)

Cases of haematuria have occurred in the course of the administration of thymol for ankylostome infection, especially in anaemic subjects and those who have suffered frequently from malaria. Investigations have led the author to conclude that (1) Thymol has a very strong haemolytic action; (2) the normal blood serum has a marked protective power, due to the presence of sulphates and sulphuric acid which render the thymol harmless by combining with it; (3) the resisting power of the red blood corpuscles of patients with ankylostomes is not much below that of the healthy person, while it is slightly weaker in patients suffering from ankylostomes along with an enlarged spleen from chronic malaria; (4) The protective power against thymol haemolysis in ankylostome infections shows hardly any difference from that of the normal serum.

R. T. L.

**GIUDICE.** Sur un Cas d'Appendicite à Ankylostomes.—*Ann. d'Hyg. et Méd. Colon.* 1913. April-May-June. Vol. 16. No. 2. pp. 436-440.

The author gives a detailed clinical history of a case in which a perforated appendix was found to contain seven ankylostomes. Of these five female worms were fastened to the mucosa while two males were discovered under the mucous membrane in the neighbourhood of the ulceration which led to the perforation. The appendix was 6 cms. in length, supple and normal in shape; its lumen was not obstructed. There was no sign of chronic inflammation and the appendicitis is attributed to the damage done by the worms. Although a third of the cases of appendicitis are said to be due to intestinal parasites the ankylostome has not hitherto been associated with the condition. [The species of ankylostome is not stated and no details regarding the worms are given. It is possible that the parasites are oesophagostomes, for which the large intestine and especially the caecum is the normal habitat, *vide* THOMAS's case from Brazil.]

R. T. L.

## ASCARIDIASIS.

LE ROY DES BARRES. Note sur un Cas d'Ascariodiose hépatique.—  
*Bull. Soc. Méd.-Chirurg. de l'Indochine.* 1913. July.  
Vol. 4. No. 7. pp. 329-330.

Barres records an extraordinary series of symptoms extending over a period of years, which were diagnosed as due to ulceration of the stomach or possibly associated with lesions of the gall bladder, but on operation proved to be due to the presence of *Ascaris lumbricoides* in the bile duct

The patient was 36 years of age and had suffered intermittently from abdominal pain for 20 years. These pains had become almost of daily incidence during the past three years and of increasing severity, and during the last year were almost intolerable. The pain was epigastric, radiating to the flanks and to the right shoulder, and occurred about half an hour after food. Vomiting was frequently induced by the patient himself to relieve his agony. The vomit consisted of food mixed with a large amount of mucus. Haematemesis never occurred. The gastric juice showed a hyperacidity of about one third over normal, and gastric ulcer, with the possibility of a lesion of the gall bladder, was diagnosed. As the general condition became gradually worse laparotomy was performed and a small cicatrix was observed on the anterior surface of the stomach near the pylorus. The cystic duct was found to be almost obliterated while the gall bladder was distended to the dimensions of a medium sized pear. The bile duct was dilated to the thickness of a little finger and from it were evacuated two *Ascaris lumbricoides* and a calculus of the size and shape of a prune-stone. The case terminated fatally.

R. T. L.

Row (T. G. S.). Round Worms and Pregnancy.—*Indian Med. Gaz.* 1913. Oct. Vol. 48. No. 10. pp. 395-396.

On the tenth day of the puerperium, which had proceeded perfectly normally, the temperature suddenly rose to 105° F. There was constipation, the tongue was thickly furred and the lochial discharge, which was gradually lessening, turned again blood red in colour and became quite profuse. There was slight wandering at times and, except for the lochial symptoms, most of the symptoms of puerperial septicaemia began to supervene. The fever continued for six days between 103° F. and 105° F., rising in the evenings without abatement in spite of calomel, quinine and other antipyretics. Involution progressed satisfactorily and the discharge was free and odourless. An efficient dose of santonin followed by an ounce and a half of castor oil effected the discharge of about 20 round worms. The temperature almost immediately returned to normal and in a couple of days the discharge had diminished and the general condition had considerably improved. Recovery followed. As three similar cases had been encountered the routine treatment of a 5 gr. dose of santonin on the 4th night after delivery has been adopted with success by the author, santonin being an *emmenagogue* as well as an *anthelmintic*.

R. T. L.



GALLET DE SANTERRE. **Cas de Diarrhée Dysentérique d'Origine Ascaridienne.**—*Ann. d'Hyg. et Méd. Colon.* 1913. April-May-June. Vol. 16. No. 2. pp. 452-454.

In six cases admitted to the hospital at Hanoi as diarrhoea, dysenteric diarrhoea or dysentery an examination of the faeces, revealed numerous ova of *Ascaris*. After treatment with calomel, santonin and castor oil the stools became normal and the patients were dismissed as cured.

R. T. L.

#### DRACONTIASIS.

LISTON (Wm. Glen). **Report of the Bombay Bacteriological Laboratory for the Year 1912.** (Section 6. Dracontiasis, Guinea-Worm Disease. pp. 32-36.) 1913. Bombay: Printed at the Government Central Press.

The author states that Dr. TURKHUD has continued his investigations into the etiology of guineaworm disease.

The embryos of the guineaworm do not seek out the cyclops but are sought after and swallowed as food by the cyclops. The worm can be seen at first coiled up within the stomach of the intermediate host, whence it escapes into the body cavity by rupture of the gut wall. The young worm thereafter undergoes developmental changes which result in the shedding of its original skin at about the seventh day.

Cyclops containing living guineaworms have been kept alive for 53 days. Attempts to experimentally infect twelve *Macacus sinicus* were fruitless. Some were fed by stomach tube with water containing a number of living cyclops which had been infected from six to fifty-three days previously with guineaworm embryos. Again, embryos were placed upon the abraded skin and injected subcutaneously. The monkeys have now been under observation for more than a year and although some have been carefully examined post-mortem no worms have been detected.

It is observed that even in the most severely infected villages where the village animals (cats, dogs, goats, hens, and cattle) partook of the same water as the inhabitants none of these animals were ever known by the villagers to suffer from guineaworm.

Five persons, having volunteered, were given each five infected cyclops in which were one to two guineaworms fourteen days old, but sufficient time has not yet elapsed for results to manifest themselves.

At a small village called Sarsola, about ten miles from Thana, a well was found in which out of 114 cyclops examined no fewer than 44 were found naturally infected with *Dracunculus* larvae. It was learnt that it was a common practice among the boys to scramble down the steep sides and bathe in the well which contained at times little more than enough water to fill a moderately sized bath tub.

R. T. L.

O'DONOGHUE (D. F.). Case of Guineaworm.—*Jl. London School of Trop. Med.* 1913. Apr. Vol. 2. Pt. 2. p. 146.

A fluctuating tumour over the middle half of the right tibia was found, on incision, to contain reddish-looking pus and four female Guineaworms. The patient, when first seen, complained of pain and swelling in the leg, headache and vomiting. There was a temperature of 102° F. An urticarial rash extended from the hip to the ankle.

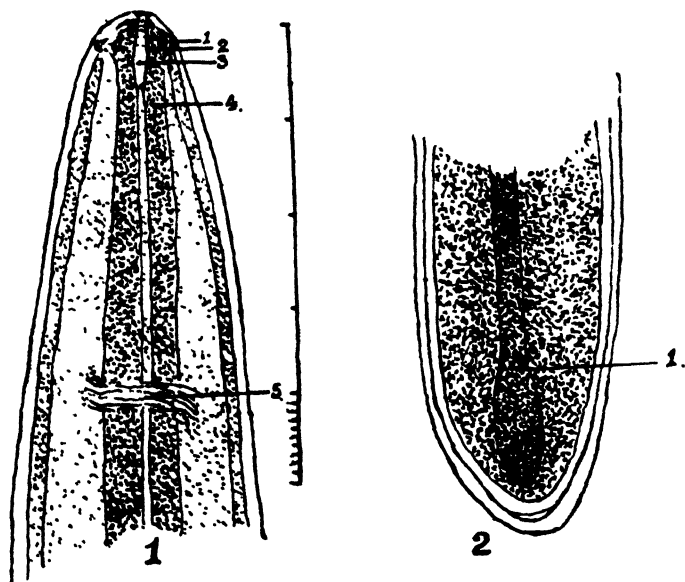
[A differential blood-count showing some eosinophilia is given. As the likelihood of an intestinal infection with helminths does not appear to have been considered the details are omitted as valueless].

R. T. L.

#### UNCLASSIFIED.

SMITH (A. J.) & DENNEY (O. E.). *Agamomermis Restiformis* (Leidy). Stiles. (P) from the Human Urethra.—*Amer. Jl. Trop. Diseases & Prev. Med.* 1913. Oct. Vol. 1. No. 4. pp. 281-287.

In 1880 LEIDY described under the name *Filaria restiformis* a blood red worm, passed alive from the urethra by a labourer in West Virginia. Among others RAILLIET regards the case as



*Agamomermis restiformis.*

Fig. 1. Anterior extremity of specimen : 1, 2, papillae ; 3, pharynx ; 4, oesophagus ; 5, neural ring.

Fig. 2. Tail end of specimen : 1, blind extremity of intestine (?).

spurious as this "Filaria" belongs to the *Mermithidae* which are parasitic only in insects.

A second case is recorded in the present paper. The worm was passed from the urethra by a man of the so called "poor white

class" in North Carolina, who had, some few days previously taken a large dose of spirits of turpentine. Before this he had complained of pain in the back, difficulty when assuming a standing posture and pain of a bearing-down character in the region of the bladder. After the worm was removed these symptoms subsided and have not returned.

The parasite is a cylindrical filariform worm almost 20 inches long and of peculiarly rigid consistence, mouth is terminal without lips but surrounded by six papillae, alimentary canal incomplete, practical absence of generative parts, peculiar diagonally striated fine markings of the skin, and Holomyarial type of muscular arrangement (see figure). There seems little doubt that the specimen is an undeveloped form, probably the second larval stage, of a *Mermis*.

R. T. L.

MCNEIL (H. L.). *An Improved Method of Extracting Ova from Stools.*—*Jl. Amer. Med. Assoc.* 1913. Nov. 1. Vol. 61. No. 18. p. 1628.

A slight modification of YAVITA's method of extracting ova from stools has given the author excellent results.

A particle of the stool about the size of a cherry is placed in a test tube; 5 cc. of a 25 per cent. mixture of antiformin is then added. This is mixed well and warmed over a flame but not boiled. Five per cent. of ether is then added and the whole well shaken. The mixture is then filtered through one layer of gauze and centrifuged for one minute by water centrifuge. Four layers are formed. The eggs are found in the lower layer. It is claimed that this method is superior to that recommended by Dock and Bass as it requires less time and less residue is thrown down with the ova. It is valuable in detecting hookworm ova in stools as the capsule of the egg is not injured in the least by the mixture.

R. T. L.

MANCEAUX (L.). *Sur les Polynucléaires Éosinophiles Hématophages.*—*Compt. Rend. Soc. Biol.* 1913. Oct. 24. Vol. 75. No. 29. pp. 240-241.

M. WEINBERG and P. SÉGUIN in a recent publication showed that the polynuclear eosinophiles were capable of digesting not only bacteria but red cells as well. These results, based upon experiments *in vitro*, are now confirmed by observations *in vivo* upon pleural exudate in two successive cases. M. WEINBERG, commenting upon the communication, adds a note that the red cells of the horse when injected into the peritoneal cavity of the guinea-pig are not only engulfed but also completely digested by the eosinophiles.

R. T. L.

## TYPHUS.

ANDERSON (John F.). **The Problem of Typhus in the United States.**  
—*Jl. Amer. Med. Assoc.* 1913. June 14. Vol. 60. No. 24.  
pp. 1845-1846.

After calling attention to the identity of Brill's Disease with typhus, the author proceeds to discuss the prevalence of typhus in the United States.

Assuming the ratio of one case of typhus to 47 of typhoid, found by LEE in the Massachusetts General Hospital (see this *Bulletin*, Vol. 2, p. 3), it is estimated, from the number of cases returned as typhoid, that during 1912 typhus was present as follows:—

New York	... 72 cases.	Chicago	... 22 cases.
Baltimore	... 22 „	Philadelphia	... 34 „
Boston	... 10 „	Washington	... 12 „

The above assumption is probably an underestimate, for at the Jewish Hospital of Brooklyn during the years 1910, 1911, and 1912 the ratio of typhus to typhoid was as high as 1 to 2.6 instead of 1 to 47.

Also in New York, during 1910, 3,735 cases of typhoid were reported to the department of health, and from Lee's ratio the number of cases of typhus would be 77. During that year 59 cases of typhus occurred in two of the hospitals, which leaves only 21 more cases to be accounted for in all the other hospitals and in private practice. In view of this it is certainly reasonable to assume that the estimated number of 77 is far below the number of cases that actually occurred in that city. Therefore although the disease has had a low case mortality and shows but little tendency to spread, it appears to be more widely distributed in the United States than has hitherto been supposed, and the possibility should ever be borne in mind that it may acquire virulence and epidemic prevalence.

E. Hindle.

HEGLER (C.) & VON PROWAZEK (St.). **Untersuchungen über Fleck-fieber. (Vorläufiger Bericht.)** [Observations on Typhus. (Preliminary Note.)] — *Berlin. Klin. Wochenschr.* 1913. Nov. 3. Vol. 50. No. 44. pp. 2035-2040.

The authors observed more than a hundred cases of typhus in the hospital at Belgrade in the early part of 1913, during the epidemic that attacked the Servian army.

Their observations are divided into two parts, the first on the clinical symptoms being by Hegler, whilst the second part, on the etiology of the disease, is by Prowazek. In the first part Dr. Hegler remarks that the epidemic was not very severe, as the mortality was only about 10 per cent., though in Belgrade it was somewhat higher. Most of the cases were in men between the ages of 20 and 30 and the majority of the patients were total abstainers. The incubation period was usually 12 to 14 days but in some cases as long as 3 weeks. In the majority of the patients the fever continued for 10 or 12 days at 39°-40° C.,

and then dropped to 35° or 36°. The patients were usually kept in hospital for another fortnight after the temperature dropped before being discharged.

In addition the author gives a detailed account of the various clinical symptoms observed, together with notes on the complications, diagnosis, and prognosis of the disease. With regard to prophylaxis great care was taken to remove all lice (body and head) from the patients and as a result there were very few cases of hospital infection, whilst in the field hospitals of Southern Servia, where these precautions were not taken, there were numerous cases of infection being acquired in the hospital.

In the second part, Prowazek gives an account of the examination of 51 cases and also experimental work with animals performed with a view to discover the causative agent of the disease. In the blood of these 51 patients examined at Belgrade the polymorpho-nuclears did not present the changes typical of many other febrile complaints. Instead the nucleus shewed signs of fragmentation and the whole leucocyte became filled with particles of chromatin. These cells, with Giemsa, stained intensely red and were found to contain numerous long or round bodies, also diplococcus forms, and frequently irregular granules in addition. At the beginning of the infection very many of the leucocytes are free from these bodies but later more of them shew this appearance. [These leucocytes seem to be identical with those referred to by RABINOWITSCH, as "Türkischen Reizformen," see below p. 643.]

The author then proceeds to discuss the nature of these intra-leucocytic bodies and inclines to the opinion that they are related to the Strongyloplasma described by LIPSCHÜTZ, that belong to the Chlamydozoa. During convalescence these bodies agglomerate in the cells and finally tend to disappear.

In addition the curious double bodies, first described by RICKETTS and WILDER, were observed in the blood of a number of cases but the author remarks that the significance of these forms is still very obscure. In the exudate of a blister (produced by a Spanish Fly Blister) polymorpho-nuclear leucocytes containing the round and long bodies were observed, but the proportion of leucocytes shewing these bodies was not greater than in the case of those in the blood. On examination of sections of the organs of typhus cases, trachoma-like bodies were observed in the endothelial cells of the heart, lung, liver, kidney, etc.

Monkeys were infected both by the injection of blood from typhus patients and also, in one case, by injecting a louse that had been removed from an infected person two days previously. Charts are given illustrating the course of the temperature in two of these monkeys. Guineapigs on which infected lice were fed did not, as would be expected, shew any rise in temperature or other signs of infection. These animals, however, were easily infected by injections of blood both from a typhus patient and an infected monkey. [The injection of lice apparently was not attempted.] In neither monkeys nor guineapigs were the typical bodies observed in the leucocytes. Infected lice were carefully examined and in one case small coccoid bodies, and also diplococcus forms were observed.

In conclusion the author gives particulars of methods for staining the small bodies occurring in the leucocytes of typhus patients.

[It is evident that further work on the etiology of typhus is much to be desired, as Prowazek does not seem to come to any definite opinions regarding the causative agent of the disease.

No mention is made of RABINOWITSCH'S *Diplobacillus*.]

E. H.

ARZI (L.) & KERL (W.). i. *Variola und Flecktyphus Studien an den bosnischen Rückwanderern aus dem Balkan*. [Studies on Smallpox and Typhus amongst the Bosnians returning from the Balkans.]—*Wiener Klin. Wochenschr.* 1913. May 15. Vol. 26. No. 20. pp. 787-795.

ii. *Ueber den Typhus exanthematicus. Beobachtungen im Seelazarett San Bartolomeo (dir. Arzt See-oberarzt Dr. M. Kaiser) gelegentlich der Flecktyphusepidemie im Frühjahr 1913*. [Typhus Fever. Notes and Observations in the Marine Hospital of St. Bartholomew during the Typhus Epidemic of the Spring of 1913.]—*Arch. f. Dermatol. u. Syphilis*. Orig. 1913. Sept. Vol. 118. No. 1. pp. 386-464. With 3 plates.

i. The authors have examined cases of both smallpox and typhus occurring in the Lazaret St. Bartholomeo, Trieste, amongst the Bosnians returning from the Balkans.

With regard to typhus it is interesting to note that, although it is generally an extremely rare disease, amongst the refugees returning from the Balkans several cases were observed and therefore the association between war and typhus seems to be still maintained.

The description of twelve of these cases is reserved for a future communication, but a detailed account is given of the clinical symptoms of two women suffering from the disease. The breaking out of haemorrhages is said to be a general and characteristic symptom, accompanied by the development of the typical exanthem.

With regard to the causative agent of the disease the authors have never been able to detect any specific micro-organisms in the blood. Moreover, employing the method described by RABINOWITSCH (this *Bulletin*, Vol. 1, p. 400) they have obtained uniformly negative results with the deviation of the complement.

ii. This is a more detailed account of the above with lengthy references to the literature on the subject and is accompanied by three coloured plates illustrating certain types of exanthem.

E. H.

LÉVÊQUE. *Note sur l'Endémie Typhique de l'Aurès: "La Fièvre de Tkout": Typhus ou Paludisme?* (Rapport des 9 Septembre 1910, 28 Novembre 1910, 3 Février 1912, et 18 Mai 1913).—*Arch. de Méd. et Pharm. Militaires*. 1913. Aug. Vol. 62. No. 7. pp. 203-204.

The station of Tkout, Aurès, founded by the French in Algeria in 1885, has always had a very bad reputation from a health point

of view, as a large proportion of all Europeans sent there succumbed to a hitherto undiagnosed fever. The author has made several visits to this region and is of the opinion that this so-called "Tkout Fever" is merely typhus. The evidences in support of this view are: (1) the almost constant presence of a petechial exanthem in the patients; (2) the contagious nature of the disease; (3) its spread by nomadic tribes in the course of their wanderings; and (4) the indisputable occurrence of typhus in the province of Aurès, which has been regarded as a centre of this disease for a very long period.

E. H.

**KULKA (Wilhelm).** *Der Flecktyphus und die gegenwärtigen Ergebnisse seiner Aetiologie und Epidemiologie.* [Typhus and the Present State of our Knowledge of its Etiology and Epidemiology.]—*Das österreich. Sanitätswesen.* 1913. Nov. 6. Vol. 25. No. 45. pp. 1505-1513.

This article consists of an excellent summary of our knowledge of the etiology and epidemiology of typhus with special reference to its occurrence in Galicia.

E. H.

**RABINOWITSCH (Marcus).** i. *Ueber den Flecktyphuserreger.* [The Etiology of Typhus.]—*München. Med. Wochenschr.* 1913. Nov. 4. Vol. 60. No. 44. pp. 2451-2452.  
ii. *Hämatologische Diagnose des Flecktyphus.* — *Deut. Med. Wochenschr.* 1913. Nov. 6. Vol. 39. No. 45. pp. 2199-2200.

i. The author recalls that in 1908 he discovered the causative agent of typhus and named the organism *Diplobacillus exanthematicus*. He then proceeds to discuss an article by P. T. MÜLLER (see below) and in addition gives some further information regarding this *Diplobacillus*.

In fresh cultures the organism practically always appears as a diplobacillus and degeneration forms only develop in old cultures. In fresh cultures the organism is always Gram-positive whilst in old ones it becomes Gram-negative.

Cultures are best obtained by using a medium consisting of the best ascitic-broth (ää) mixed with four per cent. glycerin. 100 cc. of this mixture must be injected with three to five cc. of the blood of a typhus patient obtained shortly before the crisis. After the crisis the organism seems to disappear from the blood.

Guinea-pigs can be infected both by the injection of cultures and also the blood of typhus patients. The virulence of the disease varies very much, for in some cases the incubation period in the animal was only five days, whilst in others it was as much as 37 days. When the guinea-pig develops the disease it shews a rise in temperature of  $1\frac{1}{2}$  to  $3^{\circ}$  for a period of three to eight days. During this period pure cultures of the *Diplobacillus* can be obtained from the heart-blood of the guinea-pigs, whether they have been infected by the inoculation of cultures or blood

from a human typhus patient. Moreover guinea-pigs that have been infected by injections of either cultures or typhus blood are immune against a second injection of either of them.

Cultures that have been heated for half an hour to 60° C., when injected into guinea-pigs, produce no infection; so there is every hope of being able to prepare a vaccine against this disease.

ii. The author remarks that in its early stages it is exceedingly difficult to differentiate between typhus and both typhoid fever and relapsing fever. Therefore, a more precise method of diagnosing the disease is much to be desired and the author believes that the examination of the blood will furnish the means.

In 1905 he examined the blood of 58 typhus patients. The films were stained intensely with Giemsa and in 40 of the cases peculiar very deeply stained leucocytes were observed. These leucocytes resemble lymphocytes and have a very basophile protoplasm and a round or oval nucleus situated excentrically; they are said to be identical with the so-called "Türkischen Reizformen" and often constitute 3-8 per cent. of the white cells.

The authors' conclusions are as follows:—

In the first days of the disease the number of leucocytes diminishes, then gradually rises until after the crisis after which, if there is no complication, the number gradually falls again to the normal; the reverse is the case with the red cells.

In the majority of typhus patients both in the earliest stages and also during the whole period of the disease, it is possible to detect the "Türkischen Reizformen" and these furnish a means of diagnosing the infection.

E. H.

MÜLLER (Paul Th.). **Vorläufige Mitteilung über bakteriologische Befunde bei Flecktyphus.** [Preliminary Note on the Bacteriology of Typhus.]—*München. Med. Wochenschr.* 1913. June 24. Vol. 60. No. 25. pp. 1364-1365.

The author has made bacteriological examinations of various cases of typhus occurring amongst the Bosnians in the Marine Hospital of San Bartolomeo, Trieste. In Giemsa-stained preparations, diplococci, diplobacilli, cocci and ovoid rod-shaped bodies, were found present in the blood of all the cases of typhus examined and also in four patients that no longer showed any fever. The cultural examination of the blood of eleven patients gave five positive results, in each case the development of diplobacilli in broth. Attempts to cultivate the organisms on ascitic agar succeeded only three times. The isolated cultures seem to be identical with those obtained by FRERICH from cases of typhus in Tsingtau. The organisms are non-motile rods which readily round off into coccoid forms in the various culture media. The pathogenicity of the cultured organism towards animals is only very slight. When mice were inoculated with enormous doses, they succumbed within twenty-four hours and the diplobacilli were found in every part of the body. A rabbit showed a single parasite after an incubation period of seven days. On the other hand three monkeys inoculated with the cultures remained quite normal.



According to the author there can be no doubt that this *Diplobacillus* isolated at San Bartolomeo is identical with the organism described by FUERTH, RABINOWITSCH (see this *Bulletin*, Vol. 1, p. 400), and also PREDTJETSCHENSKY. Further researches are in progress on its biological characters.

E. H.

MARKL. *Beitrag zur serologischen Diagnose des Flecktyphus.* [A Contribution to the Serum Diagnosis of Typhus.]—*Wien. Klin. Wochenschr.* 1913. July 24. Vol. 26. No. 30. pp. 1234-1235.

The author states that in spite of numerous researches the causative agent of typhus remains unknown.

With regard to the serum diagnosis NICOLLE and COMTE found that the serum of typhus patients agglutinated *Micrococcus melitensis* in dilutions of 1 in 50. The author attempted to confirm this observation but obtained uniformly negative results. The blood was obtained from ten patients between the third and ninth days of the fever, from four patients four to six days after convalescence had commenced, and finally from two post-mortems.

Both microscopic and bacteriological examinations gave entirely negative results. Moreover in spite of the statements of RICKETTS, NICOLLE, CONSEIL, KRUMWEIDE, PRATT and BULLOWA, regarding the susceptibility of monkeys and guinea-pigs, the author was unable to infect either of these animals by the intraperitoneal injection of defibrinated infected blood.

With regard to the deviation of the complement the author employed as antigen an alcoholic extract of the organs of a patient that had died of typhus eight years previously. Distinct, though slight, deviation of the complement was observed in the case of all patients infected with typhus from the third day of the fever up to four to six days afterwards. The author is therefore of the opinion that this reaction is of use for diagnostic purposes, as by means of it the disease can be recognised in its early stages.

[Markl's results should be compared with those of RABINOWITSCH (see this *Bulletin*, Vol. 1, pp. 400-401), who also obtained fixation of the complement in the serum of typhus patients when employing extracts of the organs of typhus cases as antigen].

E. H.

NEWMAN (H. H.). *A Sporadic Case of Typhus Fever.*—*Jl. Amer. Med. Assoc.* 1913. Nov. 1. Vol. 61. No. 18. pp. 1629-1630.

The author describes a typical case of typhus occurring in a twelve year old girl, who had lived on a farm in the District of Columbia. During the four months preceding the attack she had previously attended school at Baltimore.

The diagnosis of the disease was confirmed by various members of the Public Health Service including ANDERSON and GOLDBERGER and this case tends to confirm ANDERSON's view that unrecognised typhus is prevalent in various parts of America.

E. H.

## HEAT STROKE.

FISKE (Charles N.). **The Effects of Exposure to Intense Heat on the Working Organism.**—*Amer. Jl. of the Med. Sciences.* 1913. Apr. Vol. 145. No. 4. (No. 493.) pp. 565-585.

In the period 1886-1910 there are recorded 1,507 cases of disability from the effects of heat in the returns of the Navy of the United States. The number of the personnel has increased from 9,191 in 1886, to 56,721 in 1910, but there have been 20 deaths only, and 33 invalids discharged from the Service on this account during the last 35 years. Sixty-four per cent. of the cases were "heat prostration," 34 per cent. were classified "thermic fever," and 4 per cent. "insolation." While the general admission rate for this cause was under 3 per 1,000 in 1909 and 1910, that of the stokers was about 8 per 1,000.

Abstracts of the Reports of many Naval Medical Officers are given, in which are described the conditions under which the Engineering Staff worked, conditions which appear to be incompatible with the support of human life. "The men of the engineer's force after four hours of severe and arduous toil in the frightful temperature of from 140° to 150° F. would come off watch and endeavour unsuccessfully to get rest and sleep in the comparatively less trying temperature of from 90° to 100°." "The ventilation of the engine room is very unsatisfactory and temperatures are recorded from 90° to 170° F." "Steaming half-power the temperature of the starboard forward fire room reached 180° F. Three men were exhausted by the heat. On September 11, 190° F. was recorded. On the fireroom galleries the thermometer registered 205° F."—"The average temperatures of fireroom No. 1 for the year was 145·8° F. with a maximum of 204° F."—"these pump rooms registered 180° F."—"Temperatures as high as 183° F. in the fireroom are recorded, with 167° as the minimum."—"An occasional temperature of 210° F., reduced 30° by changing the location of the blowers." "Steering engine room temperatures of a 110° to 115° and humidity so great that there is no evaporation from the body."

The favourable effects consequent on the substitution of oil for coal are thus commented on:—"The temperature in the fireroom rose to 140° F., which is about 10 higher than under previous conditions when burning coal. Nevertheless in this higher temperature no case of heat prostration occurred, nor during the cruise has any member of the engineer's force succumbed to heat. It is thought that the slight amount of physical exertion required to regulate the burners, as compared with passing coal, enables the fireroom watch to withstand a greater elevation of temperature. The number of men is reduced to about one half of those required for passing coal."

The chief symptoms noted in the men who were attacked are those of collapse: pallor or lividity, drenching perspirations, weakness of both voluntary and involuntary muscles, feebleness of the action of the heart and lungs, dizziness, headache, somnolence, and stupor, painful cramps of the muscles of the abdomen,

arms and legs; as a rule neither convulsions nor fever occur. Recovery is rapid. Among predisposing causes are chronic alcoholism, overcrowding, hard muscular work, fatigue, insufficient circulation of the air with a wet-bulb thermometer at 85° to 95° F., tight clothing, and not drinking a sufficiency of water.

Increased circulation of air, and asbestos sheathing of steam pipes are important preventive measures.

C. Birt.

HILLER (Arnold). *Wesen und Behandlung des Hitzschlags*. [Nature and Treatment of Heatstroke.]—*Deut. Med. Wochenschr.* 1913. June 19. Vol. 39. No. 25. pp. 1185-1188.

This is a continuation of the author's study of heatstroke in the German Army (see this *Bulletin*, Vol. 1, p. 583). As soon as the thermometer registers 30° C. in the shade, casualties arise. He states that during the heat of summer many people faint in the street, but they recover rapidly when they are removed to a shady place. A few days later however they may complain of paraesthesiae and loss of power in certain muscles. He attributes such symptoms, and also the heat exhaustion itself, to neurasthenia.

The effects of heat on the German troops during the summer manœuvres appear in four forms: 1, exhaustion on the march; 2, the asphyxial type of heatstroke; 3, the paralytic type, and 4, the psychopathic type.

Instances of the first form are always very numerous, but as soon as the soldier is relieved of his accoutrements and rests awhile, he recovers rapidly. The asphyxial type was noted in 329 out of 470 cases of heatstroke, the mortality of this form being 7 per cent. The soldier tries to fight against the increasing prostration which is coming over him, and marches on with tottering gait, fixed stare, and cyanosed face till he falls senseless to the ground. He is apparently dead for his respiration has stopped, his pulse is imperceptible, and he is deeply cyanosed. Artificial respiration is the only remedy and must be continued for two hours in some cases.

119 of the 470 cases of heatstroke were of the paralytic type; 60.5 per cent. of which were fatal. The chief symptoms are deep coma, convulsions which recur every few minutes, vomiting and diarrhoea, and hyperpyrexia. The body temperature must be reduced by cold effusions. Venesection is required to prevent the onset of oedema of the lungs and brain, for the former condition is found in 21 per cent. of autopsies, and the latter in 12 per cent.

22 of the 470 cases were of the psychopathic form, all of which recovered. The patient generally is confused or suffers from muttering delirium with hallucinations and fleeting delusions. Sometimes he is violent and excited, and imagines that he is surrounded with enemies from whom he seeks to escape by every means in his power. LAVERAN states there were 11 suicides among 200 cases of heatstroke in one brigade during operations in Oran, Algeria.

83.6 per cent. of the German soldiers who were prostrated by heat, were men out of condition by alcoholism, obesity, sedentary occupations, or chronic diseases of the heart or lungs; extensive adhesions of the lungs were found in 36 per cent. of 47 necropsies. According to ZINIZ and SCHUMBURG 3 liters of water are lost by a man in the form of sweat during a four hour march, and with this about 20 grams of salts are excreted. If perspiration should be checked by want of water or other causes, the heat regulating mechanism of evaporation is impaired and the body temperature rises rapidly.

C. B.

PEMBREY (M. S.). **Heat-Stroke.**—*Jl. R. Army Med. Corps.* 1913. Aug. Vol. 21. No. 2. pp. 156-164.

This is an analysis of the reports of 50 cases of heatstroke which occurred among European troops in India between June, 1909, and September, 1910.

If the evaporation from the skin be sufficiently rapid, the human body can support temperatures above  $100^{\circ}\text{C}$  for a few minutes. BRADEN and FORDYCE underwent exposure to a temperature of  $115\text{--}126^{\circ}\text{C}$  in a dry atmosphere for 15 minutes without discomfort. But the body temperature rises, the pulse quickens, the perspiration is profuse, and dyspnoea and exhaustion come on when the wet bulb thermometer exceeds  $88^{\circ}$  to  $90^{\circ}\text{F}$ . ( $31^{\circ}$  to  $32^{\circ}\text{C}$ .) if the air is motionless, even though the person be stripped and at rest. If muscular work is being performed, a wet bulb temperature of  $80^{\circ}\text{F}$ . ( $26.7^{\circ}\text{C}$ .) causes distress. A movement in the air of two miles an hour permits a wet-bulb temperature of  $93^{\circ}\text{F}$ . ( $33.9^{\circ}\text{C}$ .) to be borne. As the body temperature rises, the respiratory and nitrogenous exchanges are increased, hence in heatstroke there is not only a smaller loss but also a greater production of heat.

In 25 of the cases the wet-bulb temperature of the air was  $80^{\circ}\text{F}$ . ( $26.7^{\circ}\text{C}$ .) or over; in 8 cases  $84^{\circ}\text{F}$ . ( $28.9^{\circ}\text{C}$ .) or over, and in 5 cases  $85^{\circ}\text{F}$ . ( $29.4^{\circ}\text{C}$ .) or over. The highest dry-bulb temperature was  $112^{\circ}\text{F}$ . ( $44.4^{\circ}\text{C}$ .) with a wet-bulb temperature of  $82^{\circ}\text{F}$ . ( $27.8^{\circ}\text{C}$ .). The highest record with the wet-bulb was  $86^{\circ}\text{F}$ . ( $30^{\circ}\text{C}$ .) when the dry-bulb registered  $108^{\circ}\text{F}$ . ( $42.2^{\circ}\text{C}$ .). The lowest temperature by the dry-bulb was  $86^{\circ}\text{F}$ . ( $30^{\circ}\text{C}$ .), with the wet-bulb at  $76^{\circ}\text{F}$ . ( $24.4^{\circ}\text{C}$ .); the lowest temperature by the wet-bulb was  $67.8^{\circ}\text{F}$ . ( $19.9^{\circ}\text{C}$ .) with the dry-bulb at  $94.8^{\circ}\text{F}$ . ( $34.9^{\circ}\text{C}$ .).

Muscular work entails increased combustion in the body, accompanied with greater production of heat. In the experimental marches at Aldershot the maximum rectal temperature observed was  $102.4^{\circ}\text{F}$ . ( $39.1^{\circ}\text{C}$ .) The same march of 7 miles was performed by the same men on hot and cold days. On the first occasion the dry-bulb thermometer registered  $79^{\circ}\text{F}$ , and the wet-bulb  $67.5^{\circ}\text{F}$ .; on the second,  $45^{\circ}$  and  $38^{\circ}$  were the temperatures respectively. The maximum increase in the rectal temperature was  $2.3^{\circ}\text{F}$ . in the first march and only  $1.6^{\circ}\text{F}$ . in the second. On the hot day the maximum loss of water was

2,390 gms. of which 640 gms. were retained in the clothing; and on the cold day the maximum loss of water was 555 gms. only, 40 gms being taken up by the clothes. Hence the effect of work in a hot moist and still atmosphere is to increase the temperature, pulse and loss of moisture out of proportion to the work done. In 23 of the cases muscular work in the sun was noted as a factor in the causation of heatstroke.

Adjustment of the body to its rising temperature may be disturbed by unsuitable clothing and equipment and by debility from any cause. Alcohol upsets the heat regulation mechanism. The rectal temperature of men who have recovered from alcoholic poisoning has been noted as low as  $75.2^{\circ}$  F. ( $24^{\circ}$  C.). When an alcoholic is exposed to heat his temperature rapidly ascends. Seven of the cases occurred in drinkers of which 4 were fatal; 7 in moderate drinkers, 2 ending fatally; and 7 in abstainers all of which ended in recovery except one. Malaria can act in a similar manner by lessening the power of accommodation of the body. The blood of 21 of the cases was examined for malarial parasites; they were found in 2. In 18 cases the patient had suffered from malaise for a few days before his seizure.

The mortality was 20 per cent. Twenty-seven of the men were between 20 and 25 years of age, but among them there were two deaths only. Of 10 men between the ages of 39 and 41 attacked, five succumbed. The average service in India of eight of the soldiers who died was four and a half years. Thirty of the cases occurred in Multan. The attack came on in the night in four instances only. ROGERS in his analysis of 363 cases of heatstroke among British soldiers in India found that the time of onset was the hottest period of the day in the hottest months of the year in the large majority of instances.

There was one death in 22 cases in which the pyrexia did not exceed  $107^{\circ}$  F. ( $41.7^{\circ}$  C.); and 10 out of 12 men recovered whose temperature had mounted to  $107^{\circ}$ - $108^{\circ}$  F. Five out of six survived temperatures of  $108^{\circ}$ - $109^{\circ}$  F.; and 3 out of 5,  $109^{\circ}$ - $110^{\circ}$  F. Four men with temperatures  $110^{\circ}$ - $111^{\circ}$  F. succumbed.

Absence of sweating was a symptom in several cases. The application of cold douche and enema gave good results; antipyretic drugs were useless.

Heart disease was found on post-mortem examination in one case, and cirrhosis of the liver in another.

There is no bacteriological, experimental or clinical evidence that heatstroke is a specific fever as some writers have suggested.

C. B.

**Ross (T. S.). Heat-Stroke.** [Correspondence.]—*Indian Med. Gaz.* 1913. Sept. Vol. 48. No. 9. p. 372.

Cases of cerebral malaria are frequent in which the symptoms simulate heatstroke. Two Indian soldiers dropped down senseless while marching on a very hot day; malarial parasites were present in the blood of both. One died in a short time; the other recovered without any treatment except intramuscular injections of quinine. An Indian boy lay unconscious for two days with a

temperature of 106° F.; he was restored to health by the intramuscular injection of quinine. The author has met with 6 similar examples of cerebral malaria. He states that he himself has not seen a case of heatstroke. [Being an officer of the Indian Medical Service employed as Medical Officer of Health for 8 years he would not be called upon to treat many Europeans.] He says however, "I have known cases of apoplexy occurring in typically apoplectic subjects in whom the attack was immediately determined by exposure to a high temperature in a stuffy room, or railway station or railway carriage."

C. B.

NEILSON (J. L.). **Two Cases of Thermic Fever occurring in the Fireroom of a Battleship.**—*U.S. Naval Med. Bull.* 1913. Oct. Vol. 7. No. 4. pp. 579-583.

It was reported in the daily press that in August of this year several deaths from heatstroke had occurred in the fireroom staffs of trans-Atlantic liners. The U.S.S. "Illinois," a training ship for midshipmen, was cruising near Madeira when the average temperature for 11 days was 86° F. with a high humidity. On August 9 when the temperature was 85° F. a midshipman had acted as coal passer for an hour, and had been for nearly an hour in the boiler room where the temperature was 138° F. and the ventilation defective, when he suddenly collapsed: he was unconscious, cyanosed, and pulseless; his respirations soon ceased, convulsions came on, his skin was dry, the rectal temperature was 107°·5 F. Under artificial respiration continued for about a minute, he revived, his pulse was then 130 a minute. He remained comatose for 20 minutes, and was irritable and restless for several hours. No urine was passed until the following morning: it contained a trace of albumen. Recovery was rapid.

On the same day another midshipman was employed in a fireroom the temperature of which was 128° F., when he felt giddy and unable to concentrate his thoughts, or to speak clearly, or to hear distinctly. He became excited and violent for a few moments and then fell senseless. Severe convulsions ending in deep coma ensued; his pupils were contracted to pin points, his eyelids twitched; his breathing was shallow and his pulse was weak and 160 a minute; his rectal temperature was 107° F. He remained unconscious for 4 hours, and for the remainder of the night he resented being disturbed and complained of headache. On the next day his urine was faintly albuminous, his temperature was nearly 100° F. and he was somnolent. Not till two days later did his temperature fall to normal limits, but his convalescence afterwards was speedy. In both the treatment consisted in the application of ice to the head and body, and the administration of iced water enemata. Venesection to the extent of 6 ounces was employed in the second case followed by the hypodermic injection of saline fluid.

C. B.

**Annual Report of the Sanitary Commissioner with the Government of India for 1911.—1913.** Calcutta: Superintendent Government Printing, India. [Heatstroke. pp. 12 & 76.]

In the year 1911 there were 254 cases of heatstroke, 24 of which ended fatally, among the European troops in India; 13 officers, 6 women and 13 children were also attacked, but without a fatal issue in any of them. In the Indian regiments there were only 11 cases and no deaths. In the jail population of India there were 127 cases and 34 deaths. The highest incidence in the Europeans occurred at Agra where 31·8 per thousand of strength were admitted during the year; Poonamallee came next with 18·7 per thousand; there were three deaths from this cause at Ferozepore. Fatehgarh, Benares, Jubbulpore, Jhansi, Nowshera, and Sialkot were other stations in which the incidence was high. Only 10 per thousand of troops on the march were seized: this is a considerably lower ratio than was noted in any of the above-mentioned places.

C. B.

### MOSQUITOES AND THEIR DESTRUCTION.

STANTON (A. T.). *The Anopheles of Malaya. Part 1.—Bull. Entomol. Research.* 1913. Sept. Vol. 4. Pt. 2. pp. 129-133. With 4 text-figs.

This is the first of a series of papers in which the author proposes to give the results of a detailed study of the individual species of the Malayan *Anopheles*. He lays stress on the necessity of including descriptions both of immature and mature larvae in view of the growth changes which these forms undergo. The leaflets in all the palmate hairs of a given larva are not of similar form and it is necessary in all cases to indicate the segment which bears them. Palmate hairs borne on the thorax usually differ in shape from those springing from abdominal segments. The author states:—

“The synonymy suggested for the different species is based on the study of a series of specimens, derived in most cases from a single female of the species and on careful comparison of these specimens with published descriptions. It has not been possible to examine all the original types, some of which are now non-existent and others inaccessible to the writer; it is possible therefore that this synonymy may require revision at the hands of those who have opportunity for this investigation and who are also familiar with the common variations in the species itself.

“There is no doubt that in this group of insects the number of species names has been unnecessarily increased by authors attaching exaggerated importance to characters which on further study have proved to be variable, and that the natural affinities of species have been obscured by the division of the group into a multiplicity of genera.”

The author discusses the question of synonyms and points out why errors have occurred. He then describes the ovum, immature and mature larva, and the pupa. Illustrations by R. W. BLAIR aid the text.

It is to be noted that among Malayan species the mature larva of *Anopheles tessellatus* most nearly resembles that of *Anopheles kochi*.

A. Balfour.

STRICKLAND (C.). *The Myzorhynchus Group of Anopheline Mosquitoes in Malaya.*—*Bull. Entomol. Research.* 1913. Sept. Vol. 4. Pt. 2. pp. 135-142.

The title of the paper sufficiently indicates the scope of the work undertaken and the author summarises his results as follows:—

(1) The species *sinensis*, *barbirostris*, *umbrosus*, *albotaeniatus*, *asiatica* have been considered valid.

(2) The species *vunus*, *annularis*, *minutus*, *paeditaeniatus* and *separatus* have been rejected as invalid.

(3) Certain new characters of the valid species have been noticed.

(4) The descriptions of the species have been revised.

(5) The genus *Putagiamyia* has been rejected, as also the genus *Lophocelomyia*, both having been included in the genus *Myzorhynchus*.

(6) It is suggested that the *Myzorhynchus* group forms a series sufficiently distinct from other Anophelines to make it advisable to give it a separate generic title.

In a postscript he states that he goes further than STANTON, who has been unable to satisfy himself that *separatus* is distinct from *sinensis*. Strickland thinks that he has advanced sufficient evidence to prove them identical. STANTON retains *albotaeniatus* but only pending the examination of more material. As noted above Strickland regards it as certainly a distinct species. STANTON groups all the species under *Anopheles* but Strickland considers the group so well defined from other Anophelines that he feels justified in naming it as a distinct genus. For the new characters of the valid species and the revised description the original paper must be consulted.

A. B.

BUTTRICK (P. L.). *The Effect of Tides and Rainfall on the Breeding of Salt Marsh Mosquitoes.*—*Il. Economic Entomology.* 1913. Aug. Vol. 6. No. 4. pp. 352-359.

New Haven, Connecticut, has for many years had a mosquito reputation in the summer only second to that of certain places in New Jersey. The chief culprit is the banded salt marsh mosquito (*Culex sollicitans*, Walk.). Its eggs are laid singly on the salt marsh mud and lie dormant until covered by water, either tide or rain. They then hatch in a few hours; and in from six to fifteen days, according to the temperature, the adults emerge from the pupae. These migrate or are wind blown into inhabited places where they render life miserable. Some return to the marsh or its vicinity and start the next generation.

The paper is chiefly concerned with the question of tides, their effect on the breeding habits of this mosquito, and the methods of studying them so that an oiling campaign can be conducted to the best advantage. Calculations regarding the tides are based upon the so-called "prediction" tables of the United States Coast and Geodetic Survey which are highly accurate, as is shown by means of a chart giving the actual tides and the predictions in the form of curves. The paper, though useful, is chiefly of local interest. When considering the influence of rainfall the author states that though it is a factor in the abundance of mosquitoes it does not necessarily follow that the more rain the more mosquitoes. The time and size of individual rains are, however, of great importance



in the case of *C. sollicitans* and at times rain may actually prevent breeding by causing a premature hatching of eggs when there is not enough water present for full development to take place.

A. B.

BRITTON (W. E.). *Mosquito Control Work in Connecticut in 1912.* [With Discussion.] — *Jl. Economic Entomology.* 1913. Feb. Vol. 6. No. 1. pp. 89-93.

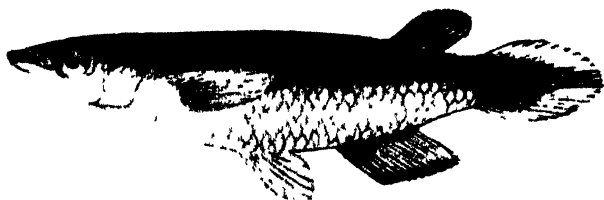
Although a complete survey of the shore region of Connecticut was made by the author's assistants in 1904 and maps and recommendations furnished each town, it was not until 1911 that anything was done. Then one community took the initiative and soon "mosquito weeks" and "donation weeks" were rife. However, despite every effort in the way of lectures and demonstrations only 5,000 dollars of the 25,000 required were forthcoming. This money was spent on draining and oiling round New Haven and, so far as the work went, the result was satisfactory. A table is given showing the cost of draining parts of the salt marshes but it scarcely merits reproduction. Kerosene and light fuel oil known as "34° distillate" were used for the petrologage. The latter, though somewhat the cheaper, proved more injurious to vegetation and to the various forms of aquatic animal life—some of which destroy mosquito larvae. It was found best to employ the large "Double Forester" pumps, made by W. B. DOUGLAS, Middletown, Connecticut, for fighting forest fires.

The author thinks the problem of salt marsh drainage and oiling is too large for communities to solve separately. The movement must be state-wide. In a discussion on the paper it was stated that the campaign at Worcester, Mass., had been highly successful, malaria which had been gaining ground having been almost entirely banished.

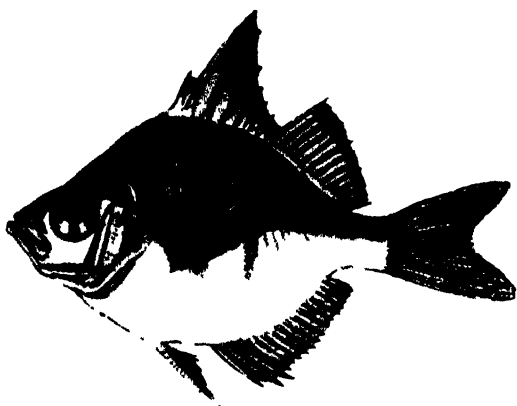
A. B.

SEWELL (R. B. S.) & CHAUDHURI (B. L.). *Indian Fish of Proved Utility as Mosquito-Destroyers.* 1912. 25 pp. With 9 text-figs. Calcutta: Sold by the Superintendent, Indian Museum. [Price 8 annas.]

The authors have compiled a useful and well illustrated pamphlet describing those genera and species of fish which in India are most likely to be of use as destroyers of mosquito larvae. It is specially intended to stimulate interest in the subject and to indicate the proper lines which such research should follow. They have found fish belonging to eight different genera to be of considerable service. In some cases, as in the genus *Haplochilus*, all the species are of equal utility; in others, however, for example *Barbus*, certain species, i.e., those which as adults do not attain a large size, are of much greater use than others. Some of the fish by reason of their shape, e.g., the flat-headed *Haplochilus*, are well suited for surface feeding in shallows, others like those belonging to the genera *Ambassis* and *Trichogaster* are compressed from side to side and deep from above downwards, thus being better adapted for somewhat deeper waters. The short notes attached to



A.



B.

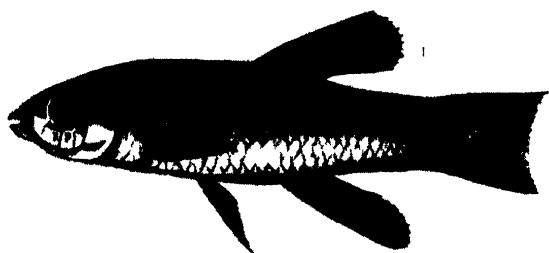


C

A. *Haplocheilichthys panchar* (nat. size).

B. *Ambassis ranua* (nat. size).

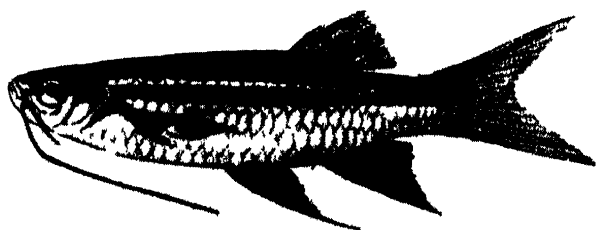
C. *Trichogaster fasciatus* (nat. size).



D.



E



F.

D. (♂) and E. (♀). *Lebias dispar* (nat. size).  
F. *Nuria danrica* (nat. size).

some of the descriptions are interesting and in addition to the fish specially mentioned a list of others of minor importance is given. It is worth noting that the value of the *Chilwa* (*Chela agentea*) as a mosquito eater has been exaggerated. Indeed it is stated not to be a surface feeder. [This is important as the *Chilwa* has been credited with "rising to the fly," and taking female mosquitoes intent on ovipositing.]

The appendix by Captain SEWELL contains general observations on pisciculture and special notes on the habits of small Indian fish. It would appear that the species most useful in mosquito reduction belong to the genera *Haplochilus*, *Ambassis*, *Trichogaster* and *Nuria*. Sewell concludes with a reference to the advantages and disadvantages of water-weeds as regards their influence on mosquito life.

[This paper will appeal to many elsewhere than in India as several of the genera mentioned are represented in other parts of the world. As the authors state a member of the genus *Labias* = *L.* or *Cyprinodon dispar* has been tested in the Anglo-Egyptian Sudan and found efficient as a larva eater, but unfortunately it proved rather difficult to transport and rear in captivity. At the present time experiments are being carried out in Khartoum with some species of *Haplochilus* obtained from Lake No on the Upper Nile. In arranging for this work the paper under review was found of value; hence it has been considered advisable to reproduce several of the illustrations.]

A. B.

GRAHAM (J. D.). *Note on Mosquito Larvae-Destroying Fish in the United Provinces.* 6 pp. folio. [1913.] Printed by F. Luker, Supdt. Govt. Press.

Major Graham has carried out a useful fish survey in certain districts of the United Provinces in India. He agrees with SEWELL that a survey of indigenous fish is of the first importance with a view to making use of any such as are available and of eradicating from tanks any highly carnivorous species as, for example, the full-grown *Aphiocephalus*. The most useful larvivorous fish found in the United Provinces seemed to be *Trichogaster fasciatus* (popular name Khasri) and *T. labius* (Bhuri), *Ambassis ranga* (Channa) and *Nuria danrica*. A variety of the last named was discovered and has been named *grahami*. Experiments were made with a considerable number of species, care being taken to reproduce natural conditions as far as possible and not to starve the fish prior to the tests. *Haplochilus* and some well-known species were not found but they probably exist somewhere in the United Provinces. There is an interesting note on the introduction of the Barbados "millions" into India. They have been studied there by the fish department of the Indian Museum and it is stated that they reproduce slowly, are inferior to *Haplochilus* as larvae-eaters, are more omnivorous and less hardy. The paper concludes with a long list of fish found in various districts of the United Provinces.

A. B.

FRIEDRICHS (K.). *Exotische und einheimische Fischarten als Vertilger der Stechmückenlarven.* [Foreign and Native Mosquito-destroying Fish.] — *Fischerei-Zeitung.* 1912. June 16. Vol. 15. No. 24. 6 pp.

The greater part of this paper is a review of the work of those who have made observations on mosquito-killing fish. Special attention is paid to a paper by NICHOLLS on the "Millions," *Girardinus poeciloides* of St. Lucia, West Indies, and to that of GRAHAM on *Haplochilus grahami* found in swamps at Lagos. This little fish belongs to the Cyprinodonts, a family which also includes the Barrigudo of Brazil (possibly *Girardinus caudimaculatus*) and other well known genera and species, for example the "Killifish" of North America (genus *Fundulus*), and the "Top Minnow," *Gambusia affinis*. The work of BENTLEY in India which deals principally with the value of *Haplochilus lineatus*, the "Piku" fish or so-called "Scooty" and of a species of *Anabas* known as "Kazari" or "Kasara" receives due notice, as does that of KING in the Anglo-Egyptian Sudan on *Ophiocephalus obscurus* and *Lebias (Cyprinodon) dispar*. Special attention is paid to the valuable investigations of SMITH\* in the United States and there is also a short description of the characteristics of the Cyprinodonts.

The author has some notes on the European sticklebacks and refers to the use of fish of economic value as larvae-eaters. In this connection he mentions the pisciculture at present practised in the rice fields of North Italy (see this *Bulletin*, Vol. 2, pp. 329-330) and points out that ducks, like fish, keep pools and ponds free from mosquito larvae. He has also something to say about eels which, although perhaps not so valuable for mosquito destruction as some other fish, are useful in certain places owing to their migratory habits, *e.g.*, in swampy parts where shallow pools tend to dry up. Eels leave such pools and seek other water collections, a habit which they share in common with the above-mentioned *Haplochilus grahami*. They can also live in the brackish water so often found in the neighbourhood of seaside watering places. The paper though it contains little original matter is a useful resumé, and concludes with a classification of the different genera mentioned, the table being compiled from the works of BOULENGER and GARMAN.

A. B.

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\* SMITH (J. B.). Report of the New Jersey State Agricult. Exper. Station upon the Mosquitoes occurring within the State, their Habits, Life History, etc. —1904. Fish versus Mosquito Larvae. pp. 92-113.

## BOOK REVIEW.

GRAHAM-SMITH (G. S.). *Flies in Relation to Disease. Non-blood-sucking Flies.*—xiv. + 292 pp. With 24 plates and 32 text-figs. [Vol. 1 of the Cambridge Public Health Series.] 1913. Cambridge: at the University Press. [10s. 6d. net.]

The particular merit of this useful book is that it gives, among other things, a very full and exact account of the evidence, experimental and other, upon which the general suspicion of the house-fly as an agent in the transmission of specific pathogenic organisms is justified. Entomologists for the most part have been content to hold the insect up to execration as obviously and irretrievably impure of life, but here are to be found those facts, duly marshalled and submitted to careful bacteriological scrutiny—and those measured and qualified inferences from the facts—without which the vehement denunciations of the entomologist must be open to reactionary criticism.

As one of the declared objects of the book is to assist those who may have to formulate plans of research, the chapter on the ways whereby house-flies carry and distribute *Bacillus prodigiosus* must be singled out as a model example of the methods by which the great fly-problem, in all its specific ramifications, is to be attacked. In this "standard" chapter the author describes in great detail a series of experiments devised to determine the persistence of the bacillus on and in the fly, the possibility of germination in the fly's crop, the length of time that infective faeces, etc., are deposited by the fly, the influence of different kinds of food on the infectivity of the faeces, the manner and measure of infection of different media by infective flies, and the possibility of infecting clean flies from the excreta of those already contaminated. Other necessary aspects of the problem are focussed, with a precision that will greatly facilitate the labours of future investigators, in the chapter on the fate of micro-organisms ingested by the larva; in the chapter on anthrax, in which experiments to determine the exact resistance of spores, both in the adult fly and in the larva, and also in the dead fly, are described; and in the chapter on epidemic diarrhoea, in which the bearings of epidemiological evidence are carefully checked. In this connexion also the short chapter on the methods of keeping flies under observation in captivity may be of some use. The author also does good service in pointing out all the qualifications that have to be made in the application of inferences drawn from experimental manipulations of captive flies and cultures, to the doings of free flies in an ordinary environment.

In dealing historically with flies as disseminators of specific *noxæ*, the author sets out to review, as far as possible, the potentialities of the insect as defined by experiment; the equipoise between the appearance of the insect and the out-break of the particular disease, as revealed by statistics; and the evidence of the existence of the specific *noxæ* in free flies. The diseases severally reviewed are typhoid, dysentery, paratyphoid, epidemic diarrhoea, cholera, tuberculosis, anthrax, diphtheria, ophthalmia, plague, staphylococcal infection, poliomyelitis, small-pox, tropical sore, trypanosomiasis, yaws, and parasitic-worm-infections. Naturally there must here be great inequality of historic treatment; but in the short section on bacillary dysentery it is surprising to find the admirable work done by P. H. BAER in Fiji completely overlooked.

The entomology of the house-fly—its structure, life-history, habits, etc., and the external features that distinguish it from other species of flies commonly found in houses—is considered in much detail; but as this part of the book, though of great general interest, has no special connexion with problems peculiar to tropical medicine, it hardly comes within the scope of this journal. The same remarks apply to the chapters on the diseases of flies, and the prevention and control of house-flies.

The short section on Myiasis is below the critical standard of the rest of the book: the very interesting observations and experiments of ROUBAUD on *Auchmeromyia* and *Cordylobia* are not recorded, and *Bengalia depressa* receives an undue share of consideration.

The bibliography is copious and without doubt will be extremely useful: personally I feel the greatest admiration for Dr. Graham-Smith's courage in undertaking it. But why, since Government Reports are included in it, is there no mention of those of Colonel H. B. THORNHILL of the Indian Staff Corps—an enlightened officer who, as a cantonment magistrate in Bengal, started a thoroughly scientific campaign against the house-fly more than twenty years ago?

There is an omission in the first line of the Preface that will never please, particularly as the whole paragraph has little pertinence to a book on flies that do not suck blood. Had Dr. Graham-Smith not read a paper\* in Vol. XIV., 1879, of the Journal of the Linnean Society, Zoology, when he wrote this paragraph?

A. Alcock.

\* MANSON (Patrick). On the Development of *Filaria sanguinis hominis*, and on the Mosquito considered as a Nurse. *Jl. Linnean Soc., Zoology*. 1879. Vol. 14. pp. 304-311.—Ed.

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## LIST OF REFERENCES.

Compiled by R. L. SHEPPARD, Librarian.

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**Amoebiasis (including Amoebic Dysentery and Liver Abscess).**

- BAERMANN (G.) & HEINEMANN (H.). Die Behandlung der Amobendysenterie mit Emetin.—*München. Med. Wochenschr.*, 1913. May 27. Vol. 60. No. 21, pp. 1132-1135; and June 3, No. 22, pp. 1210-1213.
- CARDARELLI (Antonio). Come si Diagnostica l'Ascenso epatico.—*Riforma Medica*, 1913. June 21. Vol. 29. No. 25, pp. 791-796.
- CHAUFFARD (A.). Absès Dysentérique du Foie ouvert dans les Bronches. Guérison rapide par l'Emétine.—*Rev. de Thérapeutique*, 1913. Mar. 15. Vol. 80. No. 6, pp. 181-186.
- . Grand Absès Amibien du Foie. Guérison rapide par le Traitement Chirurgical suivi de la Cure d'Emétine.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Mar. 20. (3 ser.) Vol. 29. No. 10, pp. 630-633.
- . Dysenterie Amibienne Chronique. Guérison rapide par la Cure d'Emétine.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 17. (3 ser.) Vol. 29. No. 12, pp. 753-758.
- . La Dysenterie Amibienne Chronique.—*Presse Méd.*, 1913. May 14. Vol. 21. No. 39, pp. 389-391.
- CONOR (A.). La Dysenterie Amibienne en Tunisie.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 316-317.
- COSTA (S.). Absès Amibiens du Foie, partiellement ouverts dans les Bronches et dans l'Intestin. Guérison par les Ponctions et les Injections d'Emétine.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 17. (3 ser.) Vol. 29. No. 12, pp. 746-751.
- COUTEAUD. La Chirurgie, l'Emétine et l'Ipéca dans le traitement des Absès du Foie.—*Bull. et Mém. Soc. de Chirurg. de Paris*, 1913. June 17. Vol. 39. No. 22, pp. 941-949.
- CRAIG (Charles F.). The Identity of *Entamoeba histolytica* and *Entamoeba tetragena*. A Preliminary Note.—*Jl. Amer. Med. Assoc.*, 1913. May 3. Vol. 60. No. 18, pp. 1353-1354.
- . The Relation of Parasitic Amoebae to Disease.—*United States War Dept. Office of the Surgeon General. Bulletin No. 2*, 1913. Jan., pp. 95-113.
- DARLING (S. T.). The Rectal Inoculations of Kittens as an Aid in Determining the Identity of Pathogenic Entamoebae.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 149-153.
- . Budding and other Changes described by Schaudinn for *Entamoeba histolytica* seen in a Race of *E. tetragena*.—*Jl. Amer. Med. Assoc.*, 1913. Apr. 19. Vol. 60. No. 16, p. 1220; and *Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 171-173.
- . Budding and other Forms in Trophozoites of *Entamoeba tetragena* simulating the "Spore Cyst" Forms attributed to "*Entamoeba histolytica*."—*Arch. Internal Med.*, 1913. May 15. Vol. 11. No. 5, pp. 495-506. With 3 plates.
- . The Identification of the Pathogenic Entamoeba of Panama.—*Ann. Trop. Med. & Parasit.*, 1913. June 10. Vol. 7. No. 2, pp. 321-329.
- DESSY (S.) & MAROTTA (R. A.). Contribución al Tratamiento de la Enteritis Disentérica y del Absceso del Hígado (Amibiano), con el Método de Rogers.—*Semana Médica*, 1913. Apr. 3. Vol. 20. No. 14, pp. 797-799.

- DESTÉFANO (José). Dos Casos de Disenteria tratados con el Método de Rogers.—*Semana Médica*, 1913. May 22. Vol. 20. No. 21, pp. 1189-1192.
- DOPTER. Traitement de l'Amibiase Hépatique et Intestinale par l'Éméline.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 10. (3 ser.) Vol. 29. No. 11, pp. 686-689.
- DUFOUR (Henri) & THIERS (J.). Dysenterie Chronique Amibienne traitée par le Chlorhydrate d'Éméline. Présentation de Malade.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 24. (3 ser.) Vol. 29. No. 13, pp. 827-829.
- ERDMANN (Rh.). Experimentelle Ergebnisse über die Beziehungen zwischen Fortpflanzung und Befruchtung bei Protozoen, besonders bei *Amoeba dysploidea*.—*Arch. f. Protistenkunde*, 1913. Mar. 22. Vol. 29. No. 1, pp. 84-127. With 2 plates and 3 text-figs.
- GAIDE. Le Diagnostic Différentiel des Kystes Hydatiques et des Absès du Foie. (Considérations Cliniques).—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 5-17.
- GREY (George M.). Liver-Abscess.—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Part 2, pp. 141-143.
- JAMES (W. M.). Infection with *Entamoeba tetragena*.—*New York Med. Jl.*, 1913. Apl. 5. Vol. 97. No. 14, pp. 702-705.
- JOB (E.) & LÉVY (L.). Un Cas de Dysenterie Amibienne Chronique traitée par l'Éméline.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. May 15. (3 ser.) Vol. 29. No. 16, pp. 988-993.
- LA CAVA (Francesco). La Chemioterapia della Dissenteria da Amebe. Due Casi curati col Cloridrato di Emitina secondo il Metodo di L. Rogers.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 189-197.
- LECOMTE. Carie costale consécutive aux Absès du Foie.—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 17-48.
- LOW (George C.). The Administration of Emetine by the Mouth in Amoebic Dysentery. [Memoranda].—*Brit. Med. Jl.*, 1913. June 28. pp. 1369-1370.
- LYONS (Randolph). The Treatment of Amebic Dysentery with Subcutaneous Injections of Emetine Hydrochlorid. Report of Six Cases.—*Jl. Amer. Med. Assoc.*, 1913. Apr. 19. Vol. 60. No. 16, pp. 1216-1220.
- MALLANNAH (S.). The Value of Emetine in Liver Abscess. [Memoranda].—*Brit. Med. Jl.*, 1913. June 7, pp. 1206-1207.
- MARCHEUX (E.). Le Chlorhydrate d'Éméline dans la Dysenterie Amibienne.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 313-316.
- MAXWELL (J. Preston). The Use of Emetine Salts in the Treatment of Amoebic Dysentery.—*China Med. Jl.*, 1913. Mar. Vol. 27. No. 2, pp. 116-119.
- MILIAN. Le 606 est un Spécifique puissant de la Dysenterie Amibienne et l'Éméline peut être un Médicament Antisymphilitique.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Mar. 20. (3 ser.) Vol. 29. No. 10, pp. 626-630.
- MÜLLER (O.). Die Diagnose und Behandlung des dysenterischen Leberabszesses.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. May. Vol. 17. No. 9, pp. 289-303; and No. 10, pp. 335-351. With 2 plates and 3 text-figs.
- MYER (Jesse S.) & COOK (Jerome E.). Amebic Dysentery with Special Reference to the Ipecac Treatment.—*Interstate Med. Jl.*, 1913. Mar. Vol. 20. No. 3, pp. 248-250.
- ORNSTEIN (Otto). Zur Ätiologie der Amöbenruhr.—*Arch. f. Protistenkunde*, 1913. Mar. 22. Vol. 29. No. 1, pp. 78-83. With 10 text-figs.
- PAVIOT (J.) & GARIN (Ch.). Etude sur la Dysenterie Amibienne Autochtone.—*Jl. de Physiol. et de Path. Générale*, 1913. Mar. 15. Vol. 15. No. 2, pp. 842-850.

- PERVÈS & OUDARD.** Une Série de Vingt Cas personnels d'Abscès du Foie des Pays Chauds.—*Arch. de Méd. et Pharm. Navales*, 1913. Apr. Vol. 99. No. 4, pp. 241-270; and May. No. 5, pp. 321-350.
- ROUGET.** Abscès Amibien du Foie traité par la Ponction évacuatrice et les Injections sous-cutanées d'Emétine. Guérison.—*Bulls. et Mém. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 24. (3 ser.) Vol. 29. No. 13, pp. 809-812.
- SAMBUC** (Edouard). Notes Cliniques sur les Abscès du Foie au Tonkin.—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 48-103.
- SEWELL** (E. P.). A Case of Amoebic Abscess of the Liver which had burst into the Lung, cured by Emetine Hydrochloride.—*Jl. R. Army Med. Corps*, 1913. June. Vol. 20. No. 6, pp. 700-702. With 1 chart.
- DE VERTEUIL** (Fernand L.). Note on a Case of Amoebic Dysentery of Three and a Half Years' Duration rapidly cured by Injections of Emetine Hydrochloride.—*Lancet*, 1913. June 28, p. 1803.
- See also Dysentery (B. Unclassed).*

### Beriberi.

- BARSICKOW** (Max). Experimentelle Untersuchungen über die Therapeutische Wirkung der Hefe bei der alimentären, multiplen Polyneuritis der Meerschweinchen und Tauben.—*Biochemische Zeitschr.*, 1913. Vol. 48, pp. 418-426. With 1 plate.
- COOPER** (Evelyn Ashley). The Preparation from Animal Tissues of a Substance which cures Polyneuritis in Birds induced by Diets of Polished Rice.—Part 1.—*Biochemical Jl.*, 1913. May. Vol. 7. No. 3, pp. 268-274.
- COZANET.** Relation d'une Epidémie de Bérubéri ayant sévi à Gao (Haut-Sénégal-Niger).—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 157-169.
- DOYLE** (Stanley B.). Beriberi. A South American Aspect of the Disease.—*New York Med. Jl.*, 1913. Apr. 19. Vol. 97. No. 16, pp. 828-829.
- EIJKMAN** (C.). Ueber die Ursache der Beriberikrankheit.—*München Med. Wochenschr.*, 1913. Apr. 22. Vol. 60. No. 16, pp. 871-872.
- . Ueber die Natur und Wirkungsweise der gegen experimentelle Polyneuritis wirksamen Substanzen.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. May. Vol. 17. No. 10, pp. 328-335.
- FENTON** (E. G.). Etiology of Beri-Beri. [Memoranda].—*Brit. Med. Jl.*, 1913. June 14, pp. 1271-1272.
- FUNK** (Casimir). Studies on Beri-Beri. Further Facts concerning the Chemistry of the Vitamine-Fraction from Yeast.—*Brit. Med. Jl.*, 1913. Apr. 19, p. 814. With 1 plate.
- . Studies on Beri-Beri, VII. Chemistry of the Vitamine Fraction from Yeast and Rice Polishings.—*Jl. of Physiology*, 1913. June 19. Vol. 46. No. 3, pp. 173-179.
- GREGG** (Donald). Infantile Beriberi in the Philippines.—*Boston Med. & Surg. Jl.*, 1913. May 8. Vol. 168. No. 19, pp. 676-678.
- HIGHET** (H. Campbell). Studies on Beri-beri and its Prevention in Siam. Being a Report upon Certain Investigations on Beri-Beri carried out in Siam by the Medical Officers of the Health Department of the Ministry of Local Government. 46 pp. With 5 charts. *Government of Siam Ministry of Local Government*. Printed by order of H.E. the Minister of Local Government.
- RICHER.** Rapport sur une Epidémie de Bérubéri à la Prison de Loango (1911-1912).—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 145-156.

- SCHÜFFNER (W.). Ist die Beriberi eine auch in Europa heimische Krankheit?—*München. Med. Wochenschr.*, 1913. Mar. 25. Vol. 60. No. 12, pp. 624-647.
- STRONG (Richard P.) & CROWELL (P. C.). The Etiology of Beri-beri.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 195-198.
- TAZAWA (N.). On the Nutritive Defect caused by Rice Flour.—*Sei-i-Kwai Med. Jl.*, 1913. May 10. Vol. 32. No. 5. (Whole No. 375.) [In Japanese.]
- TSUZUKI (J.). Eine Beriberiepidemie auf Fischerbooten bei den Tsishima-Inseln (Japan).—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. May. Vol. 17. No. 9, pp. 306-308.
- VEDDER (Edward B.). The Prevention of Beriberi.—*United States War Dept. Office of the Surgeon General. Bulletin* No. 2, 1913. Jan. pp. 87-94.
- & CLARK (Elbert). A Study of Polyneuritis gallinarum. A Fifth Contribution to the Etiology of Beriberi.—*Philippine Jl. of Science. Sect. B. [Philippine Jl. Trop. Med.]*, 1912. Oct. Vol. 7. No. 5, pp. 423-458. With 11 plates.
- WIELAND (Hermann). Neuere Forschungen über die Ursache der Beriberi-krankheit.—*München. Med. Wochenschr.*, 1913. Apr. 1. Vol. 60. No. 13, pp. 706-708.
- YABE (T.). Infantile Beriberi.—*Sei-i-Kwai Med. Jl.*, 1913. May 10. Vol. 32. No. 5. (Whole No. 375.) [In Japanese.]
- YAMAGIWA (R.), KOYANO (T.), MIDORIKAWA (H.) & MOGI (T.). Experimental Study on the Cause and Nature of the Beriberi. Report I. (The Original in No. 23. Vol. 26, 1912, of the *Jl. of the Tokyo Med. Assoc.*)—*Sei-i-Kwai Med. Jl.*, 1913. Feb. 10. Vol. 32. No. 2. (Whole No. 372.) pp. 12-15.

### Blackwater Fever.

- COLES (Alfred C.). Protozoal-like Structures in the Blood in a Case of Blackwater Fever.—*Lancet*, 1913. May 3. pp. 1230-1232. With 1 text-fig.
- LOVELACE (Carl). The Etiology and Treatment of Hemoglobinuric Fever. A Report of Five Hundred and Fourteen Cases.—*Arch. Internal Med.*, 1913. June 15. Vol. 11. No. 6, pp. 674-684.
- LOW (George C.) & WENYON (C. M.). Cell Inclusions in the Leucocytes of Blackwater Fever and other Tropical Diseases.—*Jl. Trop. Med. & Hyg.*, 1913. June 2. Vol. 16. No. 11, pp. 161-163. With 1 coloured plate and 2 text-figs.
- DA MATTA (Alfredo Augusto). A Febre Biliosa Hemoglobinurica no Amazonas e o seu Tratamento pela Cecropia.—*Revista Med. de S. Paulo*, 1912. Sept. 30. Vol. 15. No. 18, pp. 357-364. With 4 charts.
- NAKAGAWA (K.). On the Blackwater Fever and its Clinical Study in Korenko, Formosa. (The Original in No. 16. Vol. 26, 1912, of the *Jl. of the Tokyo Med. Assoc.*)—*Sei-i-Kwai Med. Jl.*, 1913. Feb. 10. Vol. 32. No. 2. Whole No. 372. p. 12.
- NEWELL (A. G.). The Etiology of Blackwater Fever.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 177-181.
- SOREL. Traitement de la Fièvre Bilieuse Hémoglobinurique par les Injections et Lavages de Solutions Sucrées (Clinique d'Outre-Mer).—*Ann. d'Hyg. et Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 194-199.
- STANNUS (H. Stannus). The Etiology of Blackwater Fever.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, p. 181.
- . The Treatment of Suppression in Blackwater Fever.—*Jl. Trop. Med. & Hyg.*, 1913. May 1. Vol. 16. No. 9, p. 131-133. With 1 chart.

## Cholera.

- ALESSANDRINI (Giulio) & SAMPIETRO (Gaetano). Sulla Vitalità del Vibrione Colerigeno nel Latte e nelle Mosche.—*Ann. d'Igiene Sperimentale*, 1912. Vol. 22. (New ser.) No. 4, pp. 623-630.
- BAUJEAN (M.). Etude comparée des Actions Protéolytiques et Hémolytiques de quelques Vibrions Cholériques.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 25. Vol. 74. No. 14, pp. 799-800.
- BLOEDORN (W. A.). Report of a Case of Cholera in the U.S.S. "Helena" and Notes on a Shanghai Epidemic.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 251-252.
- CARAPELLE. Sull Identificazione del Vibrione del Colera.—*Ann. d'Igiene Sperimentale*, 1912. Vol. 22. (new ser.) No. 3, pp. 497-520.
- . Sul Colera del 1910-1911 in Sicilia e Specialmente in Palermo.—*Ann. d'Igiene Sperimentale*, 1912. Vol. 22 (new ser.). No. 3, pp. 451-495. With 14 figs. and 2 maps.
- CRASTER (C. V.). The Properties and Agglutinations of some Nonpathogenic Vibrios.—*Jl. of Infectious Diseases*, 1913. May. Vol. 12. No. 3, pp. 472-480.
- VAN DIJKEN (H. W. J.). De Behandeling der Cholerajlders in het Militair Hospitaal te Weltevreden tijdens de laatste Epidemie.—*Geneesk. Tijdschr. v. Nederlandsch-Indië*, 1913. Vol. 53. No. 1, pp. 32-52.
- FREISE (W.). Die Epidemiologie der asiatischen Cholera seit 1899. (VI. Pandemie).—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. June. Vol. 17. Beiheft 5. 81 pp. [pp. 289-365.] With tables, maps and 1 curve.
- JOB (E.). Les Procédés nouveaux de Recherche du Vibrion Cholérique chez l'Homme.—*Arch. de Méd et de Pharm. Militaires*, 1913. June. Vol. 61. No. 6, pp. 601-622.
- KRUMWIEDE JR. (Charles) & PRATT (Josephine S.). Dahlia-Agar als Unterscheidungsmittel zwischen Cholera- und anderen Vibrionen.—*Centralbl. f. Bakt. 1 Abt. Orig.*, 1913. Apr. 16. Vol. 68. Nos. 5/6, pp. 562-566.
- LOW (R. Bruce). Preliminary Statement as to Occurrences of Plague and Cholera throughout the World during 1912.—*Reports to the Local Govt. Board on Public Health & Medical Subjects*, 1913. (New Series No. 78.) pp. 22-33.
- NAAME. Sulla Cura del Colera con l'Adrenalina.—*Gazz. d. Ospedali e. d. Cliniche*, 1913. Apr. 3. Vol. 34. No. 40, p. 416.
- NIJLAND (A. H.). Weder eenige Resultaten, met het Choleravaccin verkregen.—*Geneesk. Tijdschr. v. Nederlandsch-Indië*, 1913. Vol. 53. No. 1, p. 1-12.
- PIOVESANA. Ancora della Cura del Colera con l'Adrenalina.—*Gazz. d. Ospedali e. d. Cliniche*, 1913. Apr. 17. Vol. 34. No. 46, pp. 481-482.
- PIRAS (L.). Bakteriologische Beobachtungen, die während der Cholera-epidemie zu Genua im Jahre 1911 gemacht worden sind.—*Hygien. Rundschau*, 1913. June 1. Vol. 23. No. 11, pp. 641-658.
- POTTEVIN (Henri). Toxine et Antitoxine Cholériques.—*Compt. Rend. Acad. Sciences*, 1913. May 26. Vol. 156. No. 21, pp. 1631-1633.
- ROTHKY (Karl). Ueber die Spezifität der von sensibilisierten Choleravibrionen abgesprengten Agglutinine.—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie*, 1. Teil. Orig., 1913. June 30. Vol. 18. No. 4, pp. 369-378.
- SABELLA (Pietro). La parotite quale Complicazione nel Colera a nelle Malattie atasso-adinamiche in Genere.—*Il Morgagni* (Parte 1. Archivio), 1913. Feb. Vol. 55. No. 2, pp. 44-53.

- SALIMBENI & ORTICONI.** Essais de Traitement des Porteurs sains de Vibrion Cholérique par les Lavements de Sérum Spécifique.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 306-308.
- SEBASTIANI (Antonio).** Sui Vaccini Colerici.—*Ann. d'Igiene Sperimentale*, 1912. Vol. 22. (New ser.) No. 4, pp. 569-598.
- SEGALE (Mario).** Ricerche Anatomopatologiche, Batteriologiche e Biochimiche su tre Feti di Colerose.—*Pathologica*, 1913. Apr. 1. Vol. 5. No. 106, pp. 220-204.
- STOKES (Wm. Royal) & HACHTEL (Frank W.).** The Use of a Modified Hesse's Medium for Isolating the Typhoid Bacillus and the Cholera Spirillum from Stools.—*Centralbl. f. Bakt.* 1. Abt. Orig., 1913. June 4. Vol. 69. No. 4, pp. 346-349.
- WHYTE (G. Duncan).** The Treatment of an Epidemic of Cholera by Rogers' Method. Based on a Study of 215 Cases which required the Intravenous Infusion of Saline.—*China Med. J.*, 1913. Mar. Vol. 27. No. 2, pp. 107-116.

## Dengue.

- KHAN (Soleiman).** Dengue at Meerut. [Correspondence].—*Indian Med. Gaz.*, 1913. May. Vol. 48. No. 5, p. 204.
- SMITH (F.).** Dengue Fever among the Troops in Calcutta; its Identity with Seven-Day Fever and Three-Day Fever.—*Jl. R. Army Med. Corps*, 1913. Apr. Vol. 20. No. 4, pp. 453-458.
- STITT (E. R.).** Dengue, its History, Symptomatology, and Epidemiology.—*Bull. Johns Hopkins Hospital*, 1913. Apr. Vol. 24. No. 266, pp. 117-121. [Discussion pp. 124-125.]

## Dysentery (Bacillary and Unclassed).

### (A.) Bacillary.

- BAUGHER (Albert Howard) & GAY (Robert J.).** An Epidemic of Bacillary Dysentery in Institutional Children.—*Trans. Chicago Path. Soc.*, 1913. Feb. 1. Vol. 9. No. 1, pp. 8-10.
- BUTLER (C. S.).** Some Laboratory Notes upon the Bacillus of Dysentery.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 200-215.
- HUTT.** Neue Beiträge zur Kenntnis der Pseudodysenterie und Paradyenterie, sowie der sogenannten Mutation.—*Zeitschr. f. Hyg. & Infektionskr.*, 1913. Apr. 25. Vol. 74. No. 1, pp. 108-137.
- KRONTOWSKI (A.).** Zur Frage über die Typhus- und Dysenterieverbreitung durch Fliegen.—*Centralbl. f. Bakt.* 1. Abt. Orig., 1913. Apr. 23. Vol. 68. No. 7, pp. 586-590.
- SANGIORGI (G.) & BONGIOANNINI (G.).** Eine Bacillenruhrseuche in Piemont.—*Centralbl. f. Bakt.* 1. Abt. Orig., 1913. May 3. Vol. 69. No. 1/2, pp. 37-41.

### (B.) Unclassed.

- CANTLIE (J.).** Collosol Argentum: Its use in Sprue and Post-Dysenteric Conditions.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 15. Vol. 16. No. 8, pp. 123-124.
- DESAI (V. G.).** Dysentery.—*Indian Med. Rec.*, 1913. Apr. Vol. 33. No. 4, pp. 85-90.
- GAUDUCHEAU (A.).** Recherches sur les Dysentéries. (5e note.) Etude de quelques Actions défensives contre les Germes dysentériques.—*Bull. Soc. Méd. Chirurg. de l'Indochine*, 1913. Apr. Vol. 4. No. 4, pp. 167-177.
- LORENZ (Friedrich H.).** Zur Dysenterie der Irrenanstalten.—*Centralbl. f. Bakt.* 1. Abt. Orig., 1913. May 23. Vol. 69. No. 3, pp. 113-132.

- LUKIS** (Charles Pardey). An address on the Diagnosis and Treatment of Dysentery. Delivered before the South Midland Branch of the British Medical Association.—*Brit. Med. Jl.*, 1913. June 28. pp. 1357-1359.
- MATHIEU** (A.). Les Colites Dysentériques Graves et Hemorragiques; leur Diagnostic Différentiel et leurs Indications Opératoires.—*Jl. des Practiciens*, 1913. Apr. 12. Vol. 27. No. 15, pp. 225-227.
- WALKER** (Ernest Linwood). Quantitative Determination of the Balantidicidal Activity of Certain Drugs and Chemicals as a Basis for Treatment of Infections with *Balantidium coli*.—*Philippine Jl. of Science*. Sec. B, Trop. Med., 1913. Feb. Vol. 8. No. 1, pp. 1-15.

### Fevers (Unclassed).

- BRUCE** (W. J.). The Nature of Zambesi Fever.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 173-177.
- GABBI** (U.). La Fièvre Boutonneuse a Tripoli.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 65-68.
- . Sulle "Febbre non classificate" a Tripoli.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 172-179.

### Filariasis.

- FULLEBORN** (F.). Beiträge zur Morphologie und Differentialdiagnose der Mikrofilarien.—*Beihefte zum Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Jan. Vol. 17. Beiheft 1, pp. 7-72. With 8 plates.
- KING** (Arthur). A Case of Filariasis in Devonshire.—*Brit. Med. Jl.*, 1913. May 24. p. 1108.
- KULZ** (L.). Der tropische Muskelabszess (Myositis purulenta tropica).—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1912. May. Vol. 16. No. 10, pp. 313-324.
- LOW** (George C.). *Filaria loa* Cases: Continuation Reports.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 15. Vol. 16. No. 8, pp. 118-120.
- MEINHOF** (Heinrich). Zur Klinik und Morphologie der *Filaria* und *Mikrofilaria loa* (diurna). *Beihefte zum Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Feb. Vol. 17. Beiheft 2, pp. 5-58. With 8 text-figs.

### Elephantiasis.

- JACOB**. Varices Lymphatiques intradermiques de la Région Inguino-crurale.—*Bull. et Mem. Soc. Chirurgie de Paris*, 1913. Apr. 22. Vol. 39. No. 14, pp. 606-610.
- MÜLLER**. Zur Operation der Elephantiasis der männlichen Genitalien.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. Apr. Vol. 17. No. 8, pp. 269-272.

### Dracontiasis.

- O'DONOGHUE** (D. J. F.). Case of Guinea Worm.—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Part 2. p. 146.
- ROUBAUD** (E.). Observations sur la Biologie du Ver de Guinée. Infection intestinale des Cyclops.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 281-288. With 3 figs.

### Filariasis in Animals.

- LEGER** (André). Microfilaires sanguicoles de quelques Oiseaux du Haut-Sénégal et Niger.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 359-367.



## Heat Stroke.

- FISKE (Charles N.). The Effects of Exposure to Intense Heat on the Working Organism.—*Amer. Jl. of the Med. Sciences*, 1913. Apr. Vol. 145. No. 4 (No. 493), pp. 565-585.
- HILLER (Arnold). Wesen und Behandlung des Hitzschlags.—*Deut. Med. Wochenschr.*, 1913. June 19. Vol. 39. No. 25, pp. 1185-1188.

## Helminthiasis.

### TREMATODES.

#### Distomiasis.

- HOUGHTON (Henry S.). Notes on the Life Cycle of *Clonorchis*.—*China Med. Jl.*, 1913. May. Vol. 27. No. 3, pp. 168-171.

#### Schistosomiasis.

- CONOR (A.) & ARROUX. La Bilharziose en Tunisie: le Foyer du Djerid.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 259-261.
- EDGAR (W. Harold). Yangtze Fever.—*Statistical Report of the Health of the Navy for the Year 1911*. (Appendix), pp. 167-169. 1912. London: published by H.M. Stationery Office.
- KAY (James A.). On the Development of the Bilharzia Embryo.—*Transvaal Med. Jl.*, 1913. Mar. Vol. 8. No. 8, pp. 199-205. With 2 plates.
- MIYAGAWA (Yonej.). Beziehungen zwischen Schistosomiasis japonica und der Dermatitis, unter Berücksichtigung der Methode der Auffindung von Parasiteneiern in den Faeces, und Beiträge zur Kenntnis der Schistosomum-Infektion.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. May 23. Vol. 69. No. 3, pp. 132-142.
- MIYAKAWA (Y.). On the Nutrition of the Eggs of the *Schistosomum japonicum* and the Changes which they undergo under the Actions of Various Physical and Chemical Agents. (The Original in No. 17, Vol. 26, of the *Jl. of the Tokyo Med. Assoc.*)—*Sei-i-Kwai Med. Jl.*, 1913. Feb. 10. Vol. 32. No. 2. Whole No. 372, pp. 10-11.
- ODHNER (T.). Zum natürlichen System der digenen Trematoden. V. Die Phylogenie des Bilharzia-Typus.—*Zoologischer Anzeiger*, 1912. Dec. 10. Vol. 41. No. 2, pp. 54-71.
- PFISTER (E.). Urolithiasis und Bilharziasis.—*Arch. f. Schiff- u. Trop. Hyg.*, 1913. May. Vol. 17. No. 9, pp. 309-318. With 1 text-fig.

### CESTODES.

#### Taeniasis (Intestinal).

- BETTENCOURT (A.). Sur la Fréquence relative du *Taenia solium* et du *Taenia saginata* en Portugal.—*Arquivos Inst. Bact. (amara Pestana)*, 1913. Apr. Vol. 4. No. 1, pp. 1-5.
- BLANCHARD (R.). *Bertiella satyri*, de l'Orangoutang, est aussi parasite de l'Homme.—*Bull. de l'Acad. de Méd. Paris*, 1913. 77e année. Séance du 15 Avril. 3 ser. Vol. 69. No. 14, pp. 286-296.
- HALLÉ (J.). Rapport sur une Note intitulée: "Un Cas d'Hémoptysie à répétition, due à la présence d'un Ténia dans l'Intestin," adressée à la Société par M. le Dr. Laure, d'Hyères.—*Bull. et Mém. Soc. Méd. des Hôpit. de Paris*, 1913. Apr. 24. 3 ser. Vol. 29. No. 13, pp. 829-830.
- NAUWERCK (C.). Nochmals die "Durchbohrung des Duodenums und des Pankreas durch eine Ténie."—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. June 21. Vol. 69. Nos. 5/6, pp. 434-436. With 2 coloured plates.

**Taeniasis (Somatic).**

- DÉVÉ (F.). L'Echinococcose primitive hétérotopique des Séreuses.—*Arch. de Parasit.*, 1913. Mar. 12. Vol. 15. No. 4, pp. 497-528. With 13 text-figures.
- . Les Localisations de l'Echinococcose primitive chez l'Homme. Nécessité d'une Revision des Statistiques.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 18. Vol. 74. No. 13, pp. 735-736.
- . Échinococcose primitive avec Envahissement viscéral massif chez l'Homme.—*Compt. Rend. Soc. Biol.*, 1913. Apr. Vol. 74. No. 14, pp. 781-783.
- RENDEL (C. E. Russel). Note on a Case of Primary Hydatid of Bone.—*Lancet*, 1913. Apr. 26. p. 1162.
- VERDELET (Louis). A propos de quelques Cas de Kystes hydatiques chez l'Enfant.—*Gaz. hebdomadaire des Sciences Médicales de Bordeaux*, 1913. May 4. Vol. 34. No. 18, pp. 207-210.

**NEMATODES.**

- BRAU (P.) De l'*Anguillula intestinalis* en Cochinchine et de son Diagnostic hématologique.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 262-264.
- LANE (Clayton). *Trichostrongylus colubriformis* (Giles 1892), a Human Parasite.—*Indian Med. Gaz.*, 1913. Apr. Vol. 48. No. 4, pp. 129-132. With 14 figs.
- STILES (C. W.). [Treatment of Strongyloides Infection. (In a report of the proceedings of the Helminthological Society of Washington.)]—*Science*. New Ser. 1913. Jan. 31. Vol. 37. No. 944, p. 198.

**Ankylostomiasis.**

- ASHFORD (Bailey K.). Control and Eradication of Hookworm Disease.—*United States War Dept., Office of the Surgeon-General*. Bulletin No. 2. 1913. Jan. pp. 59-71.
- . The Economic Aspects of Hookworm Disease in Porto Rico.—*United States War Dept., Office of the Surgeon-General*. Bulletin No. 2. 1913. Jan. pp. 72-86.
- BROWN (B. W.). Hookworm Disease in Southern China.—*U.S. Public Health Rep.*, 1913. Feb. 7. Vol. 28. No. 6, pp. 250-252.
- CANDIDO (G.). Anchylostomoanemia associata a Miasi intestinale.—*Ann. Med. Navale e Coloniale*, 1913. Apr. Anno 19. Vol. 1. No. 4, pp. 394-407. With 1 text-figure.
- COCKIN (R. P.). Ankylostomiasis in Grenada.—*Parasitology*, 1913. Apr. Vol. 6. No. 1, pp. 57-67.
- FERRELL (Jno. A.). Methods for the Eradication of Hookworm Disease.—*Amer. J. Public Health*, 1913. May. Vol. 3. No. 5, pp. 492-493.
- JOHNSON (L. F.). Hookworm Disease—Pathology and Diagnosis.—*Texas State J. of Med.*, 1913. Mar. Vol. 8. No. 11, pp. 303-305.
- KUENEN (W. A.). Die Ankylostomiasis bei den javanischen Auswanderern.—*Janus*, 1913. Mar. Vol. 18. No. 3, pp. 93-103.
- LANE (Clayton). *Agchylostoma ceylanicum* [Looss], a New Human Parasite.—*Indian Med. Gaz.*, 1913. June. Vol. 48. No. 6, pp. 217-218. With 4 figures.
- MALVOZ (M. E.). Dix Années de Lutte contre l'Ankylostomiasie des Mineurs.—*Bull. Acad. Royale de Méd. de Belgique*, 1913. Mar. 29. 4 Ser. Vol. 27. No. 3, pp. 264-278.
- ROCKEFELLER SANITARY COMMISSION FOR THE ERADICATION OF HOOKWORM DISEASE. Third Annual Report. (Publication No. 7.) 1912. 180 pp. Washington: Offices of the Commission.
- SAUNDEY (Robert). A Case of Ankylostomiasis in Birmingham.—*Lancet*, 1913. May 3. pp. 1223-1225.

SCHÜFFNER (W.). Bemerkungen über die Ankylostomiasis in Niederländisch-Indien und den Wert einiger Wurmmittel.—*Janus*, 1913. Jan.-Feb. Vol. 18. Nos. 1/2, pp. 59-77.

### Trichinelliasis.

BITTNER (Alphonso). Hämatologische Untersuchungen an Kaninchen bei experimenteller Trichinosis nebst einem Beitrag zur Frage der Milzexstirpation.—*Folia Haematologica*, 1913. May. Vol. 15. No. 2, pp. 237-239.

HALL (M. C.). A Spurious Parasite reported as Trichinella.—*Science*. New Ser. 1913. Jan. 31. Vol. 37. No. 944, pp. 197-198.

LEEN (T. F.). Trichinosis.—*Boston Med. & Surg. J.*, 1913. Apr. 24. Vol. 168. No. 17, pp. 601-609.

### Ascariasis.

ALLEN (Mary D.). *Ascaris lumbricoides* as a Complication of a Surgical Operation.—*Jl. Amer. Med. Assoc.*, 1913. June 21. Vol. 60. No. 25, pp. 1953-1954.

CINAGLIA (Raniero). Importanza patogenetica dell' elmintiasi da Ascaridi specialmente in rapporto ad Affezioni chirurgiche.—*Gaz. d'Ospedali e. d. Cliniche*, 1913. Apr. 10. Vol. 34. No. 43, pp. 450-452.

CONSORTI (Domenico). Alcune considerazioni sull' elmintiasi dei Bambini (e specialmente sull' Ascaride lombricoide).—*La Pediatria*, 1912. Nov. 30. Vol. 20. No. 11 (2nd ser., Vol. 10), pp. 855-858.

LENORMANT (Ch.). L'Ascariadiase des Voies biliaires.—*Presse Méd.*, 1913. June 14. No. 49, pp. 488-491.

ROSENTHAL (R.). Ueber Ascariadiasis der Gallenwege mit Berücksichtigung eines selbst beobachteten Falles.—*Deut. Zeitschr. f. Chirurgie*, 1913. Mar. Vol. 121. Nos. 5/6, pp. 544-559.

### Oxyuriasis.

REVILLIOD (Léon). A propos de l'Oxyuriasis.—*Rev. Méd. de la Suisse Romande*, 1913. Apr. 20. Vol. 33. No. 4, pp. 317-319.

RHEINDORF. Ueber das Vorkommen der Oxyuris vermicularis im erkrankten extirpierten Wurmfortsatz des Erwachsenen.—*Medizinische Klinik*, 1913. Apr. 20. Vol. 9. No. 16, pp. 623-628. With 2 text-figures.

### Serum and Tissue Reactions, Toxins, etc.

BEDSON (S. Philipps). Lésions des Organes à Sécrétion interne dans l'Intoxication vermineuse.—*Compt. Rend. Soc. Biol.*, 1913. May 16. Vol. 74. No. 17, pp. 994-996.

GHEDINI (G.). Dopo Sette Anni di Sieroreazioni Diagnostiche delle Elmintiasi.—*Gazz. d. Ospedali e. d. Cliniche*, 1913. Mar. 13. Vol. 34. No. 31, pp. 321-323.

WEINBERG (M.) & CUICA (A.). Recherches sur l'Anaphylaxie Hydatique expérimentale. (Première Note).—*Compt. Rend. Soc. Biol.*, 1913. May 9. Vol. 74. No. 16, pp. 958-960.

— & —. Anaphylaxie Hydatique expérimentale. (Deuxième note.) Analogie des Symptômes de l'Anaphylaxie Expérimentale avec les Accidents observés chez l'Homme.—*Compt. Rend. Soc. Biol.*, 1913. May 16. Vol. 74. No. 17, pp. 987-990.

— & SÉGUIN (P.). Quelques Observations sur la Toxine Ascaridienne. Dissociation des Effets produits: Neutralisation de l'Action Toxique par le Sérum de Chevaux spontanément immunisés.—*Compt. Rend. Soc. Biol.*, 1913. May 2. Vol. 74. No. 15, pp. 855-857.

- WEINBERG (M.) & SÉGUIN (P.). Recherches sur l'Eosinophile et l'Eosinophilie. (Deuxième note.) Explication de l'Abaissement considérable du Taux de l'Eosinophilie après l'Opération du Kyste Hydatique.—*Compt. Rend. Soc. Biol.*, 1913. May 30. Vol. 74. No. 19, pp. 1096-1098.

## GENERAL AND UNCLASSIFIED.

- BERNARD (P. Noël) & KOUN (L.). Parasitisme intestinal en Annam.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 343-346.
- MAXWELL (James L.). Final Research Report for the Triennial Meeting.—*China Med. Jl.*, 1913. Mar. Vol. 27. No. 2, pp. 102-104.
- MILLOUS (P.). Helminthiase Intestinale à Thanh-hoa.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. Mar. Vol. 4. No. 3, pp. 157-158.
- RANSOM (B. H.). The Life History of *Haemonema muscae* (Carter), a Parasite of the Horse transmitted by the House Fly.—*U.S. Dept. of Agriculture. Bureau of Animal Industry. Bulletin* 163. Issued Apr. 3, 1913. 36 pp. With 41 text-figs.
- SNELL (J. A.). Report of Feces examination of 424 Cases in the Surgical Service of the Soochow Hospital. [Final Research Report.]—*China Med. Jl.*, 1913. Mar. Vol. 27. No. 2, p. 105.
- STILES (Ch. Wardell). Soil Pollution. The Chain Gang as a Possible Disseminator of Intestinal Parasites and Infections.—*U.S. Public Health Rep.*, 1913. May 23. Vol. 28. No. 21, pp. 985-986.
- WARD (Henry B.). Means for the Accurate Determination of Human Intestinal Parasites.—*Illinois Med. Jl.*, 1912. Oct. 18 pp. With 46 text-figures.

## Kala Azar (and Tropical Sore).

- ARCHIBALD (R. G.). An Interesting Case of Kala-Azar.—*Jl. R. Army Med. Corps*, 1913. May. Vol. 20. No. 5, pp. 512-521. With 1 plate.
- BASILE (Carlo). La Trasmissione sperimentale della Leishmaniosi naturale del Cane ai Topi, Conigli e Cavia.—*Atti d. R. Accad. d. Lincei. Rendiconti.*, 1913. Mar. 16. Vol. 22 (1° semest.). No. 6, pp. 392-393.
- . La Trasmissione sperimentale della Leishmaniosi del Mediterraneo ai Topi per mezzo delle Pulci.—*Atti d. R. Accad. d. Lincei. Rendiconti.*, 1913. Apr. 6. Vol. 22 (1° semest.). No. 7, pp. 468-470.
- . Sulla Leishmaniosi nel Cane e sull' Esistenza di Leishmania nel Midollo Spinale di Cani naturalmente infetti.—*Atti d. R. Accad. d. Lincei. Rendiconti.*, 1913. Apr. 20. Vol. 22 (1° semest.). No. 8, pp. 524-527.
- CARONIA (G.). Weiterer Beitrag zur Leishmania Anämie.—*Arch. f. Kinderheilkunde*, 1913. Feb. 20. Vol. 59. No. 5/6, pp. 321-347.
- . Sulla Guaribilità dell' Anemia da Leishmania (a proposito di 8 casi di guarigione osservati a Palermo).—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 90-96.
- CRESPIN. Leishmaniose et Paludisme chronique infantile.—*Caducée*, 1913. Apr. 5. Vol. 13. No. 7, p. 89.
- GABBI (U.). Au Sujet de l'Histoire du Kala-azar Méditerranéen.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 141-143.
- . Sulla Storia del Kala-azar del Mediterraneo.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 198-202.
- GORETTI (Girolamo). Su di Una Forma rara di Splenomegalia Contributo Anatomo-patologico.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 117-122.
- GRAY (A. C. H.). Leishmaniose naturelle du Chien à Tunis.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 165-166.
- GURKO (A. G.). Vier Fälle von Kala-Azar.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. May 20. Vol. 74. No. 2, pp. 355-368. With 9 figs.

- JEMMA (Rocco). Anemia da Leishmania.—*La Pediatria*, 1913. Jan. 31. (Published Mar. 15.) Vol. 21. No. 1. (2nd ser. Vol. 11.) pp. 1-43.
- KOHL-YAKIMOFF (Nina), YAKIMOFF (W. L.), & SCHOKHOR (N. J.). Leishmaniose canine à Taschkent.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 432-433.
- LIGNOS (Antoine). Deuxième Cas de Guérison de Kala-Azar infantile observé à Hydra.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 430-432. With 1 chart.
- LOMBARDO (Giacomo). Contributo allo Studio delle Alterazioni Anatomiche dell'Anemia da Leishmania.—*Pathologica*, 1913. May 15. Vol. 5. No. 109, pp. 292-296.
- PETRONE (G. A.). Un Caso di Guarigione di Anemia da Leishmania.—*La Pediatria*, 1912. Nov. 30. Vol. 20. No. 11. (2nd ser. Vol. 10.) pp. 852-854.
- ROUX (F.). Arsenic in the Treatment of Kala-Azar.—*Indian Med. Gaz.*, 1913. Apr. Vol. 48. No. 4, pp. 132-133.
- SALVATORE (Domenico). Un Caso di Kala-Azar a Derna.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 73-76.
- SCORDO. A Proposito di Alcuni Tentativi d'Infezione delle "Anopheles" con Succo Splenico di Malattie di Leishmaniosi Interna.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 84-89. With 1 plate.
- (Francesco). Die Leukocyten des Meerschweinchens und des Kaninchens in Kontakt mit den Flagellatenformen der *Leishmania Donovanii* in vitro und im Körper der Tiere.—*Centralbl. f. Bakt.* 1. Abt. Orig., 1913. May 3. Vol. 69. No. 1/2, pp. 85-89. With 1 plate.
- SPAGNOLIO (G.). Nota Clinica su alcuni recenti Casi di Leishmaniosi Interna (Kala-azar).—*Malaria e Malat. dei Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 80-82; and *Riforma Medica*, 1913. May 17. Vol. 29. No. 20, pp. 536-538.
- Leishmaniosi Canina e Umana e Loro rapporti.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 203-204.
- TOMASELLI (A.). Le Complicazioni della Leishmaniosi infantile.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 180-181.
- Tropical Sore (Dermal Leishmaniasis).**
- BATES (L. B.). Leishmaniosis (Oriental Sore) of the Nasal Mucosa.—*Jl. Amer. Med. Assoc.*, 1913. Mar. 22. Vol. 60. No. 12, p. 898.
- ESCOMEL. Première découverte de *Leishmania tropica* flagellée dans le Corps Humain. (Correspondance).—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 237-238.
- FRANCHINI (G.). Sur un Cas de Leishmaniose américaine. Le premier Cas en Italie avec la Constatacion du Parasite.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 219-226.
- KEELAN (R. S.). A Case of Bagdad Sores, showing an apparently long Incubation Period. (Mirror).—*Indian Med. Gaz.*, 1913. Apr. Vol. 48. No. 4, p. 146.
- MIGONE (L. E.). La Buba du Paraguay, Leishmaniose Américaine.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 210-218. With 4 figs.
- VERROTTI (G.). Un Nuovo Caso (6°) di Boubas brasiliana o Malattia del Breda.—*Giorn. Italiano d. Malattie Veneree e d. Pelle*, 1913. Mar. 28. Vol. 54. (Anno 48) No. 1, pp. 112-114.
- WENYON (C. M.). A Further Note on a Case of Dermal Leishmaniasis from S. America, with the Results of Inoculation Experiments. (Successful Inoculation of a Cat).—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Part 2, pp. 117-119.

## Leprosy.

- BARDÉZIEUX (G.).** La Prophylaxie de la Lèpre au Tonkin (Réponse à quelques Objections et Critiques).—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. Mar. Vol. 4. No. 3, pp. 88-97.
- BAYON (H.).** Report by the Government Research Bacteriologist (Leprosy) on the Necessity or Advisability of Segregation in Relation to the Conditions and Spread of Leprosy in South Africa at the Present Time; the Measures to be provided for the Prevention and Cure of Leprosy; and the Suitability of Robben Island as a Place of Detention for Lepers.—*S. African Med. Rec.*, 1913. May 24. Vol. 11. No. 10, pp. 187-194.
- BREDA (Achille).** La Lepra del Globo Oculare e dei suoi annessi.—*Gior. Ital. d. Malatt. Venere e d. Pelle*, 1913. May 10. Vol. 54. (Anno 48) No. 2, pp. 214-255.
- DUVAL (Charles W.) & HARRIS (William H.).** Further Studies upon the Leprosy Bacillus. Its Cultivation and Differentiation from other Acid-fast Species.—*Jl. of Med. Research*, 1913. May. Vol. 28. (new ser. Vol. 23). No. 1. (Whole No. 137) pp. 165-198.
- DYER (Isadore).** Aspecto Sociológico de la Lepra y la Cuestión de su Aislamiento.—*Nemana Médica*, 1913. Mar. 6. Vol. 20. No. 999. (No. 10), pp. 596-600.
- EICHMULLER (G.).** Réflexions à propos de Deux Cas de Lèpre observés à Tunis.—*Lepra*, 1913. Apr. Vol. 14. No. 1, pp. 35-39.
- FRASER (Henry).** The Cultivation of the Bacillus of Leprosy.—*Jl. Trop. Med. & Hyg.*, 1913. June 2. Vol. 16. No. 11, p. 164.
- GAUCHER.** Fréquence de la Lèpre sur la Côte d'Azur.—*Bull. Soc. Française de Dermatol. et Syphiligraph*, 1913. Apr. Vol. 24. No. 4, pp. 186-187.
- & **BOINET.** Traitement de la Lèpre par les Injections Intraveineuses de Salvarsan et les Piqures d'Abeilles.—*Bull. Soc. Française de Dermatol. et Syphiligraph*, 1913. Mar. Vol. 24. No. 3, pp. 172-174.
- JEANSELME (E.).** A propos de la Communication de MM. Brocq et Pomaret sur un Nouveau Produit injectable pour le Traitement de la Lèpre.—*Bull. Soc. Française de Dermatol. et Syphiligraph*, 1913. Mar. Vol. 24. No. 3, pp. 149-150.
- KUPFFER (A.).** Die Lepra in Estland.—*Lepra*, 1913. Apr. Vol. 14. No. 1, pp. 14-32.
- LOMBARDO (C.).** Singolari formazioni di Elastina entro cellule giganti in Casi di Lepra.—*Giorn. Italiano d. Malattie Veneree e d. Pelle*, 1913. Mar. 28. Vol. 54. (Anno 48) No. 1, pp. 75-79.
- McCoy (George W.).** Care of Lepers in Hawaii.—*U.S. Public Health Rep.*, 1913. Apr. 18. Vol. 28. No. 16, pp. 726-728.
- MARCHOUX (E.).** Etiologie et Prophylaxie de la Lèpre.—*Bull. Soc. Française de Dermatol. et de Syphiligraph.*, 1913. May. Vol. 24. No. 5, pp. 247-253.
- MEULLENGRACHT (E.).** Communication concernant la Lèpre en Serbie.—*Lepra*, 1913. Apr. Vol. 14. No. 1, pp. 33-34.
- MÖLLERS (B.).** Serologische Untersuchungen bei Leprösen.—*Deut. Med. Wochenschr.*, 1913. Mar. 27. Vol. 39. No. 13, pp. 595-596.
- MONTESANTOS (Denis).** Un Coup d'Oeil sur la Question de la Contagiosité et l'Hérédité de la Lèpre.—*Presse Méd. d'Egypte*, 1913. Apr. 1. Vol. 5. No. 7, pp. 105-106.
- MORROW (Robert).** History of Leprosy in South Africa up to the Establishment of the First Leper Asylum, Hemel en Aarde.—*S. African Med. Rec.*, 1913. May 24. Vol. 11. No. 10, pp. 174-183.
- MURRAY (C. F. K.).** The Administrative Side of the Leprosy Question.—*S. African Med. Rec.*, 1913. May 24. Vol. 11. No. 10, pp. 183-187.

- PAPELLIER (E.).** Das Luetiker Bad Kusatsu in Japan.—*München. Med. Wochenschr.*, 1912. July 30. Vol. 59. No. 31, pp. 1718-1719.
- PEIPER (Otto).** Die Bekämpfung der Lepra in Deutsch-Ostafrika. Auf Grund amtlichen Materials bearbeitet.—*Beihefte z. Arch. f. Schiffsu. Trop.-Hyg.*, 1913. May. Vol. 17. Beiheft 4, 105 pp. [183-233]. With 34 text-figures, 3 plates, and 2 maps.
- SERRA (Alberto).** Lo stato attuale della Lepra in Sardegna.—(*Clinica Dermosifilopatica della Regia Università di Cagliari.*), 1912. 28 pp. With 2 plates. Cagliari: Prem. Stab. Tipogr. Ditta G. Dessi.
- . La Séro-Réaction de Wassermann chez les Lapins inoculés de Lèpre à la Chambre Antérieure de l'Oeil.—*Lepra*, 1912. Vol. 12. No. 3, pp. 139-146.
- . Di un Raro Particolare Strutturale del Sistema Venoso Parenchimale nelle Capsule Surrenali di un Leproso.—*Pathologica*, 1913. June 15. Vol. 5. No. 111, pp. 347-351.
- . Alcuni casi di Sifilide, Lepra e Psoriasi trattati col "606".—*Gior. Ital. d. Malatt. Veneree e d. Pelle*, 1913. May 10. Vol. 54. (Anno 48) No. 2, pp. 182-213.
- SOREL (F.).** Recherche du Bacille de Hansen dans les Ganglions de Personnes Saines vivant dans l'Entourage des Lépreux.—*Bull. Soc. Path. Exot.*, 1912. Nov. Vol. 5. No. 9, pp. 698-702.
- THOMPSON (J. Ashburton).** Experimental Leprosy: a Perspective.—*Lepra*, 1913. Apr. Vol. 14. No. 1, pp. 1-13.
- VERROTTI (G.).** Risultati ottenuti dalle Inoculazioni intraperitoneali di Emulsione di Leproma nei Conigli.—*Giorn. Italiano d. Malattie Veneree e d. Pelle*, 1913. Mar. 28. Vol. 54. (Anno 48) No. 1, pp. 82-91. With 1 coloured plate.
- ZAMBACO.** La Lèpre dans l'Égypte actuelle.—*Presse Méd. d'Égypte*, 1913. Mar. 15. Vol. 5. No. 6, pp. 89-92.

## Malaria.

- ATKINSON (J. M.).** A Clinical Test for Malarial Fever.—*Lancet*, 1913. June 28. pp. 1802-1803.
- BATES (John Pelham).** A Review of a Clinical Study of Malaria Fever in Panama.—*Jl. Trop. Med. & Hyg.*, 1913. May 15. Vol. 16. No. 10, pp. 145-153, and June 16. No. 12, pp. 177-184.
- BEACH (T. B.) & LEESON (H. H.).** An Outbreak of Malaria in "F" Company, 2nd Devon Regiment.—*Jl. R. Army Med. Corps*, 1913. Apr. Vol. 20. No. 4, pp. 450-452.
- BILLET (A.).** Action de la Quinine sur les Hématozoaires du Paludisme.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 336-339.
- BLANCHARD (R.) & LANGERON (M.).** Le Paludisme des Macaques (*Plasmodium cynomolgi* Mayer, 1907).—*Arch. de Parasit.*, 1913. Mar. 12. Vol. 15. No. 4, pp. 529-542. With 2 plates.
- & ———. Nouvelles Recherches sur le Paludisme des Macaques d'après les Notes Posthumes de Xavier Bouniol.—*Arch. de Parasit.*, 1913. Mar. 12. Vol. 15. No. 4, pp. 599-607. With 3 coloured plates.
- BOOGHER (Leland).** Malarial Hematuria.—*New York Med. Jl.*, 1913. June 21. Vol. 47. No. 25. (Whole No. 1803), pp. 1291-1293.
- BOUILLIEZ (M.).** Nouvelles Recherches expérimentales sur un Plasmodium des Singes.—*Compt. Rend. Soc. Biol.*, 1913. May 23. Vol. 74. No. 18, pp. 1070-1072.
- BRIGNONE (Emiliano).** La Propaganda e Profilassi Antimalarica nelle scuole comunali di Terranova Monferrato durante l'anno 1913. Relazione a S. E. il Ministro dell'Istruzione.—*Propaganda Antimalarica*, 1913. June. Vol. 6. No. 3, pp. 57-63.

- BRÜNN (W.) & GOLDBERG. Das Cisternenproblem bei der Bekämpfung der Malaria in Jerusalem.—*Berlin Klin. Wochenschr.*, 1913. Apr. 7. Vol. 50. No. 14, pp. 639-640.
- CACACE (Ernesto). Educazione Antimalarica e Profilassi Antimalarica Scolastica in Italia nel 1911.—*Propaganda Antimalarica*, 1912. Dec. Vol. 5. No. 6, pp. 131-136.
- CANTIERI (Collatino). Le Sieroreazioni per il Tifo (Vidal), Paratifi, Bacterium Coli e Melitense (Wright) col Siero di Sangue Malarico.—*Riv. Crit. di Clin. Med.*, 1913. Mar. 15. Vol. 14. No. 11, pp. 161-168.
- CELLI (Angelo). La Malaria in Italia durante il 1910. Ricerche Epidemiologiche e Profilattiche (Concluded).—*Propaganda Antimalarica*, 1912. Dec. Vol. 5. No. 6, pp. 121-130.
- . La Malaria in Italia durante il 1911. Ricerche Epidemiologiche e Profilattiche.—*Ann. d'Igiene Sperimentale*, 1913. Vol. 23. (new ser.). No. 1, pp. 1-61.
- . La Malaria in Italia durante il 1911. Ricerche Epidemiologiche e Profilattiche.—*Propaganda Antimalarica*, 1913. Apr. Vol. 6. No. 2, pp. 25-35; and June. No. 3, pp. 49-56.
- . Die Malaria in Italien im Jahre, 1912. 14. Jahresbericht.—*Centralbl. f. Bakt.*, 1. Abt., Referate, 1913. June 5. Vol. 57. No. 12/13, pp. 353-376.
- CHUNDRA (J. L.). Malaria and its Prophylaxis.—*Indian Med. Rec.*, 1913. Jan. Vol. 33. No. 1, pp. 1-5.
- CORVINO (Raffaele). Lotta Antimalarica in Cancellò ed Arnone.—*Propaganda Antimalarica*, 1912. Dec. 31. Vol. 5. No. 6, pp. 136-139.
- CRESPIN. Leishmaniose et Paludisme chronique infantile.—*Caducée*, 1913. Apr. 5. Vol. 13. No. 7, p. 89.
- EYSELL (Adolf). Spinne und Stechmücke.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. June. Vol. 17. No. 12, pp. 414-415.
- VON EZDORF (R. H.). Preliminary Studies of Malarial Fevers in Alabama.—*Southern Med. J.*, 1913. Apr. Vol. 6. No. 4, pp. 226-229.
- . Malarial Fevers in Alabama. A Study of the Prevalence and Geographic Distribution of the Disease throughout the State during the Calendar Year 1912.—*U.S. Public Health Rep.*, 1913. Apr. 4. Vol. 28. No. 14, pp. 641-644. With map.
- FAZZARI (G. B.). Malaria ed Epatite Suppurata.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 77-79.
- FRAGALE (V.). Contributo allo Studio della Malaria nei neonati e nella prima infanzia.—*Gaz. Internaz. di Med. Chir. Igiene*, 1913. Mar. 22. No. 12, pp. 265-267.
- FRÓES (João A. G.). Un Caso de Aphemia Transitoria Palustre. (Aphasia Motora Verbal, Funcional, de Origem Paludica).—Reprint from *Brazil-Médico*, 1912. [41 pp. Rio de Janeiro: Lyp. Bernard Frères.]
- GURKO (A. G.) & HAMBURGER (J.). Zur Frage über die Kultur des Plasmodiums der tropischen Malaria nach Bass und Johns. Vorläufige Mitteilung.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. May 20. Vol. 74. No. 2, pp. 248-252.
- HAMMOND (F. A. L.). Malarial Gangrene [Mirror].—*Indian Med. Gaz.*, 1913. June. Vol. 48. No. 6, p. 228.
- HARRISON (Wm. B.). The Hypodermatic Administration of Quinine: Its Indications and Contra-indications.—*Mississippi Med. Monthly*, 1913. Mar. Vol. 17. No. 11, pp. 217-219.
- HERMANT. Fonctionnement du Service de Vente de la Quinine d'Etat dans la Province de Nghé-An en 1912.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. Apr. Vol. 4. No. 4, pp. 231-233.
- HERMS (William B.). Malaria. Cause and Control. xi + 163 pp. With 30 text figs, 1913. New York: The Macmillan Company.



- JAMAICA.** Annual Report of the Malaria Commission for the Year ended 31st March, 1912. 38 pp. With 2 charts. 1912. Jamaica: Government Printing Office, Kingston.
- JAMES (W. M.).** Notes on the Etiology of Relapse in Malarial Infections.—*Jl. of Infectious Diseases*, 1913. May. Vol. 12. No. 3, pp. 277-325. With 1 coloured plate.
- . The Canal Zone Treatment of Malaria. [Correspondence.]—*Southern Med. Jl.*, 1913. May. Vol. 6. No. 5, pp. 347-349.
- KAUFMANN (J. B.).** Account of an Outbreak of Malaria on the U.S.S. "Tacoma" resultant upon a Visit to Tampico, Mexico.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 301-302.
- KIRK (William Redin).** The Early Recognition of Tuberculosis with Especial Reference to its Confusion with Malaria.—*Southern Med. Jl.*, 1913. May. Vol. 6. No. 5, pp. 300-303.
- KNAB (Frederick).** Spider's Web and Malaria.—*Jl. Trop. Med. & Hyg.*, 1913. May 1. Vol. 16. No. 9, pp. 133-134.
- LEGER (Marcel).** Le Paludisme en Corse. Recherches Microbiologiques. Etudes Prophylactiques.—*Publication de l'Institut Pasteur*, 1913. 60 pp. (Laval: L. Barnéoud & Cie, Imprimeurs.)
- MALTESE (Paolo).** Educazione Antimalarica e Profilassi Antimalarica Scolastica nella Provincia di Trapani.—*Propaganda Antimalarica*, 1913. Apr. Vol. 6. No. 2, pp. 36-45.
- MEREU (Francesco).** La Malaria in Nurra. Condizioni Sociali, Economiche ed Igieniche.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 182-188.
- MORALES (Manuel).** Nota Documentada de Fisiopatologia general. La Anafilaxia Quinínica y su Tratamiento.—*Semana Médica*, 1913. Mar. 20. Vol. 20. No. 12, pp. 717-719.
- MÜHLENS.** Bericht über eine Malariaexpedition nach Jerusalem.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. May 3. Vol. 69. Nos. 1/2, pp. 41-85. With 6 plates and 5 text-figures.
- NICHOLLS (Lucius).** Cirrhosis of the Liver of Malarial Origin.—*Jl. Trop. Med. & Hyg.*, 1913. June 2. Vol. 16. No. 11, pp. 164-165. With 1 plate.
- O'CONNELL (Matthew D.).** The Meteorology of Malaria.—*Jl. Trop. Med. & Hyg.*, 1913. June 2. Vol. 16. No. 11, pp. 165-166.
- PARISI (Ferdinando).** Su di un Caso di Pneumonia da Malaria.—*Gazz. d. Ospedali e d. Cliniche*, 1913. May 29. Vol. 34. No. 64, pp. 671-672.
- VAN POOLE (G. M.).** A Case of Quinine Idiosyncrasy.—*Military Surgeon*, 1913. Feb. Vol. 32. No. 2, p. 192.
- REED (E. U.).** A Case of Malaria treated with Salvarsan.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 255-256.
- RIEUX (J.).** Mode d'Action de la Quinine sur *Plasmodium vivax* (var. *magna* du Prof. Laveran) de la Tierce et de la Double-Tierce bénigne de Rechute.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 153-156.
- ROSS (Ronald).** Malaria Prevention in Greece. [Correspondence.]—*Brit. Med. Jl.*, 1913. May 31, p. 1186.
- ROSSI (Giacomo).** Sul Bonificazione definitivo della Piana di Fondi e Monte San Biagio.—*Propaganda Antimalarica*, 1913. Jan.-Feb. Vol. 6. No. 1, pp. 1-17.
- SEHRWALD (Ernst).** Zur Geschichte der Malaria-Uebertragung.—*München Med. Wochenschr.*, 1913. May 13. Vol. 60. No. 19, p. 1040.
- SERGEANT (Edmond & Etienne).** Etudes Epidémiologiques et Prophylactiques du Paludisme. Neuvième et Dixième Campagnes en Algérie, en 1910 et 1911.—*Ann. Inst. Pasteur*, 1913. May 25. Vol. 27. No. 5, pp. 373-390.

- SERGI (Antonio).** La Malaria nella Riviera Ionica Reggio-Gerace nel 1911.—*Propaganda Antimalarica*, 1913. Jan.-Feb. Vol. 6. No. 1, pp. 17-19.
- . La Profilassi Scolastica Antimalarica nel 1912 in Palizzi Marina.—*Propaganda Antimalarica*, 1913. June. Vol. 6. No. 3, p. 69.
- SKELTON (D. S.).** Report on the Measures Necessary to reduce Malarial Fever in Zanzibar.—*Public Health Office, Zanzibar*, 1913. Feb.
- SOUTHERN MEDICAL ASSOCIATION.** Preliminary Report of the Commission of the Southern Medical Association for the Study and Prevention of Malaria, Year ending Nov. 4, 1912.—*Southern Med. J.*, 1913. Apr. Vol. 6. No. 4, pp. 219-226.
- THOMSON (John Gordon).** A Demonstration on the Cultivation of the Malarial Parasites (*Plasmodium falciparum* and *Plasmodium vivax*).—*Trans. Soc. Trop. Med. & Hyg.*, 1913. May. Vol. 6. No. 6, pp. 216-219. With 1 plate.
- & **THOMSON (David).** The Cultivation of One Generation of Benign Tertian Malarial Parasites (*Plasmodium vivax*) in Vitro, by Bass's Method.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Vol. 7. No. 1, pp. 153-164. With a plate by H. B. Fantham.
- TIMPANO (Pietro).** Profilassi Antimalarica nelle Scuole Elementari di Bova Marittima nel Biennio 1911-12.—*Propaganda Antimalarica*, 1913. Jan.-Feb. Vol. 6. No. 1, p. 20.
- DE VILLA (S.).** Tremore essenziale da Malaria.—*Gaz. Internaz. di Med. Chir. Igiene*, 1913. May 10. No. 19, pp. 443-444.
- WILSON (F. E.).** Note on Three Cases of Splenic Abscess occurring in So-called "Malarial Cachexia."—*Lancet*, 1913. Apr. 26. p. 1162.
- ZIEMANN (H.).** Über die Kultur der Malaria parasiten und der Piroplasmen (*Piroplasma canis*) in vitro.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. June. Vol. 17. No. 11, pp. 361-391. With 2 coloured plates and 2 curves.
- . On the Culture of Malarial Parasites and *Piroplasma canis*.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. May. Vol. 6. No. 6, pp. 220-227.

### Myiasis.

- BALZER, DANTIN, & LANDESMANN.** Un Cas de Myiase rampante due à l'*Hypoderma bovis*.—*Bull. Soc. Française de Dermatol. et de Syphiligraph.*, 1913. Apr. Vol. 24. No. 4, pp. 219-226.
- CANDIDO (G.).** Anchilostomoanemia associata a Miasi intestinale.—*Ann. Med. Navale e Coloniale*, 1913. Apr. Anno 19. Vol. 1. No. 4, pp. 394-407. With 1 text-figure.
- EDGAR (C. L.).** A Case of Screw-Worm in the Nose.—*Texas State J. of Med.*, 1913. May. Vol. 9. No. 1, p. 21.
- HECKENROTH (F.) & BLANCHARD (M.).** Note sur la Présence et l'Endémicité d'une Myiase furonculaire au Congo français.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 350-351.
- SUROUF (Jacques).** La Transmission du Ver macaque par un Moustique.—*Compt. Rend. Acad. Sci.*, 1913. May 18. Vol. 156. No. 18, pp. 1406-1408. With 2 figures.

### Pappataci Fever.

- CASTRO (A.).** Sulla Febbre dei Tre Giorni a Milazzo.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 68-73.
- KING (Harold H.).** On the Bionomics of the Sandflies (*Phlebotomus*) of Tokar, Anglo-Egyptian Sudan.—*Bull. Entomol. Res.*, 1913. May. Vol. 4. Pt. 1, p. 83-4.

- DOS SANTOS (L. Pereira). Contribução para o Estudo da "Febbre de Papatasii" em Portugal.—*A Medicina Contemporanea*, 1913. Jan. 19. Vol. 31. No. 3, pp. 20-23. With 1 text-figure and 3 curves.
- SARLO-BISOGNI (Fr.). Di un' Epidemia di "Febbre dei Tre Giorni" a Francica (Provincia di Catanzaro).—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 82-84.
- SMITH (F.). Dengue Fever among the Troops in Calcutta; its Identity with Seven-Day Fever and Three-Day Fever.—*Jl. R. Army Med. Corps*, 1913. Apr. Vol. 20. No. 4, pp. 453-458.
- SUMMERS (Sophia L. M.). A Synopsis of the Genus *Phlebotomus*.—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Pt. 2, pp. 104-116. With 2 text-figures.

## Pellagra.

- ADLER (Herman M.). The Experimental Production of Lesions resembling Pellagra.—*Boston Med. & Surgical Jl.*, 1913. Mar. 27. Vol. 163. No. 13, pp. 454-456.
- AGOSTINI. La Pellagra nell'Umbria in rapporto alle nuove vedute patogenetiche della Pellagra (XIV. Congresso della Società Freniatria Italiana).—*Rivista Sperimentale di Freniatria e Med. Legale d. Alienazioni Mentali*, 1913. Jan. Vol. 38. No. 4, pp. 264-266.
- ALESSANDRINI (G.) & SCALA (A.). Contributo nuovo alla Etiologia e Patogenesi della Pellagra.—*Polichinico*, sez. pratica., 1913. June 8. Vol. 20. No. 33, pp. 805-812.
- BOX (Charles R.). Pellagra.—*Practitioner*, 1913. June. Vol. 90. No. 6 (No. 540), pp. 940-951.
- & MOTT (F. W.). Fatal Pellagra in two English Boys, with the Results of the Pathological Investigation of one Case.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 149-156. With 2 plates.
- BRAULT (J.). Note sur la Pellagre en Algérie.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 198-201.
- DEARMAN (W. A.). Pellagra induced in a Monkey. Preliminary Report.—*Mississippi Med. Monthly*, 1913. Mar. Vol. 17. No. 11, pp. 220-221.
- DRISCOLL (T. Latane). A Theory of the Etiology of Pellagra.—*Southern Med. Jl.*, 1913. June. Vol. 6. No. 6, pp. 400-401.
- GRIMM (R. M.). Pellagra: Some Facts in its Epidemiology.—*Jl. Amer. Med. Assoc.*, 1913. May 10. Vol. 60. No. 19, pp. 1423-1427.
- HARRIS (William H.). The Experimental Production of Pellagra in the Monkey by a Berkefeld Filtrate derived from Human Lesions. A Preliminary Note.—*Jl. Amer. Med. Assoc.*, 1913. June 21. Vol. 60. No. 25, pp. 1984-1950. With 2 figures.
- HILLMAN (O. S.). Some Hematological Findings in Pellagra.—*Amer. Jl. of the Med. Sciences*, 1913. Apr. Vol. 145. No. 4 (No. 493), pp. 507-513.
- KLEIMINGER. Neue Beiträge zur Pellagralehre.—*Zeitschr. f. d. gesamte Neurolog. u. Psychiat. Orig.*, 1913. May 31. Vol. 16. No. 5, pp. 586-668.
- MACNEAL (W. J.). Observations on the Intestinal Bacteria in Pellagra.—*Amer. Jl. of the Med. Sciences*, 1913. June. Vol. 145. No. 6 (No. 495), pp. 801-806.
- MILLS (H. P.). Pellagra with Special Reference to Pathology of Gastro-Intestinal Tract.—*Jl. Amer. Med. Assoc.*, 1913. Mar. 22. Vol. 60. No. 12, pp. 889-892. With 3 figures.
- MOTT (F. W.). The Histological Changes in the Nervous System of Dr. Box's Case of Pellagra, compared with Changes found in a Case of Pellagra dying in the Abassieh Asylum, Cairo.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 157-160.

- MYERS** (Victor C.) & **FINE** (Morris S.). Metabolism in Pellagra.—*Amer. Jl. of the Med. Sciences*, 1913. May. Vol. 145. No. 5 (No. 494), pp. 705-720.
- PEARSON** (R. W. J.). Report of Egyptian Case.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 161-163.
- VON PROBLIZER** (Guido). Praktische Bemerkungen zur Diagnose der pellagrösen Hautveränderungen.—*Dermatol. Wochenschr.*, 1913. June 7. Vol. 56. No. 23, pp. 637-649.
- REED** (Howard S.). The Effect of *Diplodia Zeae* and some other Fungi upon some Phosphorus Compounds of Maize.—*New York Med. Jl.*, 1913. Mar. 22. Vol. 97. No. 12, pp. 609-611.
- SAMBON** (L. W.). The Causation of Pellagra: a Contribution to the Discussion on Dr. Sandwith's Paper.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. May. Vol. 6. No. 6, pp. 231-241.
- SANDWITH** (F. M.). Is Pellagra a Disease due to Deficiency of Nutrition?—*Trans. Soc. Trop. Med. & Hyg.*, 1913. Apr. Vol. 6. No. 5, pp. 143-148.
- SINGER** (H. Douglas) & **POLLOCK** (Lewis J.). The Histopathology of the Nervous System in Pellagra.—*Arch. Internal Med.*, 1913. June 15. Vol. 11. No. 6, pp. 565-589. With 2 coloured plates.
- TIZZONI** (G.) & **DE ANGELIS** (G.). Studien über die Biologie und die Morphologie des pleomorphen Streptobacillus der Pellagra.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. May 3. Vol. 69. Nos. 1/2, pp. 5-8.
- & —. Sul Ciclo Evolutivo dello Streptobacillo della Pellagra.—*Pathologica*, 1913. Apr. 15. Vol. 5. No. 107, pp. 225-228.
- TUCKER** (Beverly R.). Early and Undeveloped Cases of Pellagra.—*Southern Med. Jl.*, 1913. Apr. Vol. 6. No. 4, pp. 232-234.
- VOLPINO**. Ricerche sulla Pellagra.—*Giorn. R. Accad. Med. di Torino*, 1913. Jan.-Feb. Vol. 76. No. 1-2, p. 42.
- WOOD** (Edward Jenner). A Treatise on Pellagra for the General Practitioner, 1912. New York & London: D. Appleton & Co. xiv+377 pp. With 38 illustrations in text.
- . Some Problems in the Etiology of Pellagra.—*Interstate Med. Jl.*, 1913. May. Vol. 20. No. 5, pp. 437-442. With a map.

## Plague.

- AUMANN**. Erfahrungen bei einigen in das Hamburger Staatsgebiet eingeschleppten Fällen von menschlicher Pesterkrankung.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. June 21. Vol. 69. Nos. 5/6, pp. 353-378. With 5 text-figures.
- BACOT** (A. W.). The Bionomics of the Rat-Flea. [Correspondence].—*Brit. Med. Jl.*, 1913. June 14. p. 1299.
- BARBER** (M. A.). The Susceptibility of Cockroaches to Plague Bacilli inoculated into the Body Cavity.—*Philippine Jl. Science*, Sect. B. [Philippine Jl. Trop. Med.], 1912. Dec. Vol. 7. No. 6, pp. 521-524.
- BERDNIKOW** (A. I.). Einige neue Ergebnisse über die Epidemiologie der Pest. Untersuchungen der Nagetiere der Astrachanschen Steppe.—*Centralbl. f. Bakt.*, Orig., 1913. June 4. Vol. 69. No. 4, pp. 251-259.
- BROWN** (B. W.). Plague. A Note on the History of the Disease in Hongkong.—*U.S. Public Health Rep.*, 1913. Mar. 21. Vol. 28. No. 12, pp. 551-557.
- CONNOR** (F. Powell). The Results obtained in a few Cases of Bubonic Plague by the Intravenous Injections of Dilute Solution of Iodine.—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Pt. 2, pp. 148-149.

- CORRELL (R. H.). Plague Eradication in Porto Rico.—*Jl. Amer. Med. Assoc.*, 1913. May 17. Vol. 60. No. 20, pp. 1526-1527.
- . Outbreak and Suppression of Plague in Porto Rico. An Account of the Course of the Epidemic and the Measures employed for its Suppression by the United States Public Health Service.—*U.S. Public Health Rep.*, 1913. May 30. Vol. 28. No. 22, pp. 1050-1070. With 5 figs: and June 6. No. 23, pp. 1121-1149. With 3 plates.
- FOY (F. A.). Port Health Inspection as a Factor in Plague Prevention.—*Jl. of State Medicine*, 1913. May. Vol. 21. No. 5, pp. 282-289.
- GALLI-VALERIO (B.). Bacterium pseudopestis murium, n. sp.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Mar. 1. Vol. 68. No. 2, pp. 188-194. With 5 figures.
- HARKER (W. E.). Outbreak of Plague amongst Apprentices on board the British Steamer "Bellaisa."—*Annual Report of the Medical Officer of Health, Tyne Port Sanitary Authority*, 1912. 1913, pp. 24-26.
- HOSSACK (D. C.). Some Lessons of the Manchurian Plague Epidemic.—*Jl. of State Medicine*, 1913. Apr. Vol. 21. No. 4, pp. 228-233.
- ILVENTO (Arcangelo). Hygienische Beobachtungen über Ratten und Pestprophylaxe im Hafen von Palermo (1906-1910).—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. June. Vol. 17. No. 12, pp. 404-413.
- DE JONGE (G. W. Kiewiet). Antwoord aan Dr. de Raadt. (See RAADT: De critiek van Dr. Kiewiet de Jonge op mijn oordeel over het pestvaccin).—*Geneesk. Tijdschr. v. Nederlandsch-Indië*, 1913. Vol. 53. No. 1, pp. 165-172.
- LOW (R. Bruce). Preliminary Statement as to Occurrences of Plague and Cholera throughout the World during 1912.—*Reports to the Local Govt. Board on Public Health & Medical Subjects*, 1913. (New Series, No. 78), pp. 22-33.
- NUTTALL (George H. F.) & STRICKLAND (C.). Report on Rat-Fleas in Cambridgeshire.—*Parasitology*, 1913. Apr. Vol. 6. No. 1, pp. 18-19.
- . Observations on British Rat-Fleas. July-October, 1911.—*Parasitology*, 1913. Apr. Vol. 6. No. 1, p. 1.
- DE RAADT (O. L. E.). De critiek van Dr. Kiewiet de Jonge op mijn oordeel over het pestvaccin.—*Geneesk. Tijdschr. v. Nederlandsch-Indië*, 1913. Vol. 53. No. 1, pp. 155-164.
- ROBIN (L. V. E.). Conclusions générales des Délérations de la Conférence Internationale de la Peste à Moukden (Avril, 1911).—*Arch. de Med. et Pharm. Navales*, 1913. Mar. Vol. 99. No. 3, pp. 208-217; Apr. No. 4, pp. 285-293; and May. No. 5, pp. 361-374.
- SIGNORELLI (E.). Sulle Alterazioni Anatomico-Patologiche che il Bacillo della Peste o la Sua Tossina produce nei Polmoni.—*Lo Sperimentale*, 1913. May 23. Vol. 67. No. 2, pp. 155-166. With 1 plate.
- & CALDAROLA (P.). Ricerche di Agglutinazione con differenti Razze di Bacilli della Peste.—*Ann. d'Igiene Sperimentale*, 1912. Vol. 22 (New Ser.). No. 4, pp. 555-567.
- SIMPSON (Friench). Rat Proofing. Its Practical Application in the Construction or Repair of Dwellings or other Buildings.—*U.S. Public Health Reports*, 1913. Apr. 11. Vol. 28. No. 15, pp. 679-687. With 20 figures.
- STRICKLAND (C.). The Bionomics of the Rat-Flea.—*Brit. Med. Jl.*, 1913. May 31. p. 1160.
- & MERRIMAN (G.). Report on Rat-Fleas in Suffolk and North Essex.—*Parasitology*, 1913. Apr. Vol. 6. No. 1, pp. 2-18.

- SWELLENGREBEL (N. H.).** Mededeeling omtrent Onderzoekingen over de biologie van ratten en vlooiën en over andere onderwerpen, die betrekking hebben op de epidemiologie der Pest op Oost-Java.—*Geneesk. Tijdschr. v. Nederlandsch-Indie*, 1913. Vol. 53. No. 1, pp. 53-154.
- TODD (P. J.).** A Study of Plague. [Concluded].—*China Med. Jl.*, 1913. May. Vol. 27. No. 3, pp. 158-167.

### Relapsing Fever (and Spirochaetosis).

- BRONFENBRENNER (J.) & NOGUCHI (H.).** On the Resistance of Various Spirochaetes in Cultures to the Action of Chemical and Physical Agents.—*Jl. of Pharmacology & Experimental Therapeutics*, 1913. Mar. Vol. 4. No. 4, pp. 333-339.
- CHAMBERS (Helen).** A New Spirochaeta found in Human Blood. Preliminary Communication.—*Lancet*, 1913. June 21. pp. 1728-1729. With 5 text-figures. [See also Correspondence in *Lancet*, June 28, pp. 1825-1826; and July 12, p. 102.]
- GERBER.** Die bisherigen Erfahrungen mit der Salvarsan- und Neosalvarsanbehandlung der lokalen Spirochätosen.—*München. Med. Wochenschr.*, 1913. Mar. 25. Vol. 60. No. 12, pp. 634-636.
- GIEMSA (G.).** Beitrag zur Chemotherapie der Spirochätosen.—*München. Med. Wochenschr.*, 1913. May 27. Vol. 60. No. 21, pp. 1074-1078.
- GLEITSMANN.** Ueber die Beziehungen der Borrelien (Spirochäten) zu den Wirtszellen.—*Centralb. f. Bakt.*, 1. Abt. Orig., 1913. Apr. 16. Vol. 68. Nos. 5/6, pp. 493-497. With 1 plate.
- GROSS (J.).** Sporenbildung bei *Cristispira*.—*Arch. f. Protistenkunde*, 1913. Apr. 19. Vol. 29. No. 2, pp. 279-292. With 1 plate.
- HELLMANN (G.).** Ueber die im Excretionsorgan der Ascidien der Gattung *Caesira* (*Molgula*) vorkommenden Spirochäten: *Spirochaeta Caesirae septentrionalis*, n. sp. und *Spirochaeta Caesirae retortiformis* n. sp. Erste Mitteilung.—*Arch. f. Protistenkunde*, 1913. Mar. 22. Vol. 29. No. 1, pp. 22-38. With 28 text-figures.
- HIDAKA (S.).** Zur Frage der Beziehungen zwischen Syphilis- und Recurrens-Immunität.—*Zeitschr. f. Immunitätsforsch. u. experiment. Therapie.*, 1913. Apr. 14. Vol. 17. No. 4, pp. 443-448.
- JUKES (A. M.).** Spirillar Fever in the Darjeeling District, 1912.—*Indian Med. Gaz.*, 1913. June. Vol. 48. No. 6, pp. 222-225.
- KLEINE (F. K.) & ECKARD (B.).** Über die Lokalisation der Spirochäten in der Rückfallfieberzecke (*Ornithodoros moubata*).—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. May 20. Vol. 74. No. 2, pp. 389-394.
- LAMOUREUX (A.).** Présence d'*Ornithodoros moubata* dans un Foyer de Fièvre Récurrente à la Côte ouest de Madagascar.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 146-149. With map.
- LAUNOY (L.) & LÉVY-BRUHL (M.).** Les Variations Numériques et Morphologiques des Globules Blancs chez les Poules infectées de *Spirochaeta gallinarum*.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 18. Vol. 74. No. 13, pp. 754-756.
- MCCULLOCH (H. D.).** The New Spirochaete of the Human Blood and its bearing on Pathology.—*Med. Press*, 1913. May 28. New Series. Vol. 95, pp. 575-576.
- NAKANO (H.).** Ueber Immunisierungsversuche mit Spirochaeten-Reinkulturen.—*Arch. f. Dermatol. u. Syphilis. Orig.*, 1913. Mar. Vol. 116. No. 1, pp. 265-280.
- NICOLLE (Charles) & BLAIZOT (L.).** Deuxième Note sur la Courte Durée de l'Immunité dans la Fièvre Récurrente expérimentale.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 242-243.
- & — & CONSEIL (E.). Etiologie de la Fièvre Récurrente. Son Mode de Transmission par les Poux.—*Ann. Institut Pasteur*, 1913. Mar. 25. Vol. 27. No. 3, pp. 204-225.

- RIZZUTTI (G.). Osservazioni sul Tifo ricorrente a Tripoli.—*Malaria e Malat. d. Paesi Caldi*, 1913. Apr.-May. Vol. 4. No. 3, pp. 153-172.
- SCHUBERG (A.) & BÖING (W.). Ueber den Weg der Infektion bei Trypanosomen- und Spirochätenerkrankungen.—*Deut. Med. Wochenschr.*, 1913. May 8. Vol. 39. No. 19, pp. 877-879, and *Centralbl. f. Bakt.*, 1 Abt., Ref., 1913. June 14. Vol. 57. No. 14/22, pp. 226\*-230\*.
- TODD (John L.). A Note on the Transmission of Spirochaetes.—*Proc. Soc. for. Experim. Biology & Med.*, 1913. Apr. Vol. 10. No. 4, pp. 184-185. 87 (783).
- UHLENHUTH (Paul) & EMMERICH (Emil). Ueber das Verhalten des Kaninchens bei experimenteller Trypanosomen- und Spirochäteninfektion.—*Deut. Med. Wochenschr.*, 1913. Apr. 3. Vol. 39. No. 14, pp. 642-644.
- WITTROCK (O.). Beitrag zur Biologie der Spirochaeta des Rückfallfiebers.—*Zeitschr. f. Hyg. & Infektionskr.*, 1913. Apr. Vol. 74. No. 1, pp. 55-60.

### Skin, Tropical Diseases of the.

- ASSMY & KYRITZ. Ueber Salvarsanbehandlung geschwürriger Prozesse, welche durch die Vincentsche Symbiose veranlasst sind.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. Apr. Vol. 17. No. 7, pp. 217-230. With 2 plates and 1 text-figure.
- de BEURMANN & GOUGEROT. L'État actuel de la Question des Mycoses. [Rapport présenté au VIIe Congrès International de Dermatologie et de Syphiligraphie, Rome. Avril 1912].—Supplément à la *Biologie Médicale*. 69 pp. With 18 text-figures.
- FLU (P. C.). Een atypisch Geval van Mycetoma pedis, gecompliceerd met een Blastomykotische infectie.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1912. Vol. 52. No. 6, pp. 703-747. With 1 plate.
- De FREITAS (Octavio). Alastrim.—*Rev. Med. de S. Paulo*, 1912. Oct. 15. Vol. 15. No. 19, pp. 378-380.
- GOUGEROT (H.). Oosporoses ou Nocardoses Cutanées. Synonymie. Nocardoses, Oosporoses, Discomycoses, Micromycoses, Microsiphonoses, Actinomycoses, Streptothricoses.—*Gaz. des Hôpitaux Civils et Militaires*, 1913. Jan. 25. Vol. 86. No. 10, pp. 149-158; and Feb. 1. No. 13, pp. 197-204.
- GRINDON (Joseph). Granuloma Inguinale Tropicum. Report of Three Cases.—*Jl. of Cutaneous Diseases including Syphilis*, 1913. Apr. Vol. 31. No. 4. (Whole No. 367), pp. 236-240. With 3 plates.
- KERNÉIS (J.), MONFORT (F.) & HECKENROTH (F.). Quelques Remarques sur le Pian au Congo français. Pian et Ulcères phagédéniques traités par le 606.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 243-247.
- MAZZOLANI (D. A.). Le Tigne, il Pian, le Piodermatosi ed altre Affezioni Cutanee curate negli Indigeni della Tripolitania.—*Riforma Medica*, 1913. Apr. 12. Vol. 29. No. 15, pp. 396-400; and Apr. 19. No. 16, pp. 425-428. With 5 figures.
- . L'Ulcera fagedenica a Tripoli.—*Policlinico*. Sez. pratica, 1913. Apr. 27. Vol. 20. No. 17, pp. 585-589; and May 4. No. 18, pp. 621-625. With 2 text-figures.
- SABELLA (Pietro). Due Casi di "Granuloma Ulceroso delle Pudende" guariti col Neo-salvarsan a Tripoli.—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 97-101; and *Policlinico* Sez. Med., 1913. May. Vol. 20. No. 5, pp. 235-240.
- . Studio Parallelo fra la Sifilide, la Framboesia e il Granuloma ulceroso delle Pudende, osservati nella Tripolitania (con Richerche Istologiche e Sperimentali).—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 102-113.

- SUTTON (Richard L.). Mycetoma in America.—*Jl. Amer. Med. Assoc.*, 1913. May 3. Vol. 60. No. 18, pp. 1339-1342.
- WIENER (Emil). Ueber einen Vibrionen befund in einem Yemengeschwür.—*Wien Klin. Wochenschr*, 1913. Apr. 24. Vol. 26. No. 17, pp. 667-669.

### Sleeping Sickness (and other Trypanosomiasis).

- ALEXEIEFF (A.). Introduction à la Révision de la Famille *Herpetomonadidae* (= *Trypanosomidae* Doflein, 1911).—*Arch. f. Protistenkunde*, 1913. May 27. Vol. 29. No. 3, pp. 313-341. With 3 text-figures.
- ANDRIEU (Raymond). Contribution à l'étude clinique des Signes et Accidents nerveux dans la Maladie du Sommeil.—*Thèse pour Doctorat en Médecine* Faculté de Médecine de Paris, 1913. No. 212, 174 pp. Paris: Imprimerie Paul Dupont.
- AUBERT (P.) & HECKENROTH (F.). L'Atoxyl dans la "Prophylaxie chimique" de la Trypanosomiasis humaine.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 184-189.
- & —. L'Arsenophénylglycine dans la Prophylaxie chimique de la Trypanosomiasis humaine.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 272-276.
- BALFOUR (Andrew). Animal Trypanosomiasis in the Lado (Western Mongolia) and Notes on Tsetse Fly Traps and on an Alleged Immune Breed of Cattle in Southern Kordofan.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Vol. 7. No. 1, pp. 113-120. With 2 plates.
- BEVAN (Lt. E. W.). Preliminary Notes on a Trypanosome causing Disease in Man and Animals in the Sebungwe District of Southern Rhodesia.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 15. Vol. 16. No. 7, pp. 113-117. With 2 plates.
- BLACKLOCK (B.). A Study of the Posterior Nuclear Forms of *Trypanosoma rhodesiense* (Stephens and Lanham) in Rats.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Vol. 7. No. 1, pp. 101-112.
- BROWN (Alexander). Native Treatment of Sleeping Sickness—A Trial.—*Jl. Trop. Med. & Hyg.*, 1913. June 2. Vol. 16. No. 11, p. 167.
- BRUCE (David), HARVEY (David), HAMERTON (A. E.), & Lady BRUCE. Morphology of Various Strains of the Trypanosome causing Disease in Man in Nyasaland. I. The Human Strain.—*Proc. Roy. Soc.*, 1913. Apr. 7. (ser. B.) Vol. 86. No. B 587, pp. 285-302.
- , —, —, & —. Morphology of Various Strains of the Trypanosome causing Disease in Nyasaland. The Wild-game Strain.—*Proc. Roy. Soc.*, 1913. June 12. (ser. B.) Vol. 86. No. B 589, pp. 394-407. With 7 charts.
- , —, —, & —. Morphology of Various Strains of the Trypanosome causing Disease in Man in Nyasaland. The Wild *Glossina morsitans* Strain.—*Proc. Roy. Soc.*, 1913. June 12. (ser. B.) Vol. 86. No. B 589, pp. 408-421. With 7 charts.
- , —, —, & —. Infectivity of *Glossina morsitans* in Nyasaland.—*Proc. Roy. Soc.*, 1913. June 12. (ser. B.) Vol. 86. No. B 589, pp. 422-426.
- , —, —, DAVEY (J. B.) & Lady BRUCE. The Trypanosomes found in the Blood of Wild Animals living in the Sleeping Sickness Area, Nyasaland.—*Proc. Roy. Soc.*, 1913. Apr. 7. (ser. B.) Vol. 86. No. B 587, pp. 269-277.
- , —, —, & —. Trypanosome Diseases of Domestic Animals in Nyasaland. II. *Trypanosoma caprae* (Kleine).—*Proc. Roy. Soc.*, 1913. Apr. 7. (ser. B.) Vol. 86. No. B 587, pp. 278-284. With 1 plate.



- BRUMPT (E.). Evolution de *Trypanosoma Lewisi*, *Duttoni*, *Nabiasi*, *Blanchardi*, chez les Puces et les Punaies. Transmission par les Déjections. Comparaison avec *T. cruzi*.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 167-171.
- . Immunité partielle dans les Infections à *Trypanosoma cruzi*, Transmission de ce Trypanosome par *Cimex rotundatus*. Rôle régulateur des Hotes intermédiaires. Passage à travers la Peau.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 172-176.
- & GONZALEZ-LUGO. Présentation d'un Réduvide du Vénézuëla, le *Rhodnius prolixus* chez lequel évolue *Trypanosoma cruzi*.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 382-383.
- CAZALBOU (L.). Observation d'un Nouveau Trypanosome chez le Lapin.—*Receuil de Méd. Vétér.*, 1913. Mar. 15. Vol. 90. No. 5, pp. 155-158.
- CHAGAS (Carlos). Thireodite Parasitaria.—*Rev. Med. de S. Paulo*, 1912. Sept. 15. Vol. 15. No. 17, pp. 337-350. With 8 figures.
- . Les Formes Nerveuses d'une Nouvelle Trypanosomiase (*Trypanosoma cruzi* inoculé par *Triatoma magista*) (Maladie de Chagas).—*Nouvelle Iconographie de la Salpêtrière*, 1913. Jan-Feb. Vol. 26. No. 1, pp. 1-9. With 7 plates.
- DARLING (S. T.). The Immunization of Large Animals to a Pathogenic Trypanosome (*Trypanosoma hippicum* (Darling)) by Means of an Avirulent Strain.—*Jl. Experimental Med.*, 1913. May 1. Vol. 17. No. 5, pp. 582-586.
- DOBELL (Clifford). Some Recent Work on Mutation in Micro-organisms. I. Mutations in Trypanosomes.—*Jl. of Genetics*, 1912. Nov. 21. Vol. 2. No. 3, pp. 201-220.
- DOFLEIN (F.). Ueber Dauerformen und Immunität beim Froschtrypanosom.—*Berichte der Naturforschenden Gesellschaft zu Freiburg i. Br.* Vol. 20. Bericht über die Sitzung am 19 Feb. 1913. Mar. 1913.
- FRY (W. B.) & RANKEN (H. S.). Further Researches on the Extrusion of Granules by Trypanosomes and on their Further Development. (With a Note on Methods by H. G. Plimmer).—*Proc. Roy. Soc.*, 1913. June 12. (ser. B.) Vol. 86. No. B 589, pp. 377-393. With 3 plates.
- HARTOCH, ROTHERMUNDT & SCHÜRMANN. Beziehungen zwischen toxischen und chemotherapeutischen Wirkungen der Antimonpräparate, im besonderen bei Dourine.—*Centralbl. f. Bakt.*, 1. Abt., Ref., 1913. June 14. Vol. 57. No. 14/22, pp. 174\*-179\*.
- HECKENROTH (F.). Réactions locales de Début dans un Nouveau Cas de Trypanosomiase humaine chez l'Européen.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 267-269.
- . Tournée Médicale effectuée sur le Congo et l'Oubangui.—*Ann. d'Hyg. et de Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 104-144.
- & BLANCHARD (M.). Transmission du *Trypanosoma gambiense* par des Moustiques (*Mansonia uniformis*).—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 442-443.
- & ———. Recherches sur les Propriétés du Sérum des Malades atteints de Trypanosomiase au Congo français.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 444-447.
- v. d. HELLEN. Ueber den Zeitpunkt des Auftretens von Rückfällen der menschlichen Trypanosomiasis nach ihrer Behandlung mit Arsenophenylglyzin.—*Arch. f. Schiff- u. Trop.-Hyg.*, 1913. Apr. Vol. 17. No. 7, pp. 280-242.
- HIRSCH (Rahel). Trypanosomen-Wärmestich-Anaphylatoxinfieber beim Kaninchen.—*Zeitschr. f. Exper. Patholog. u. Therapie.*, 1913. Apr. 24. Vol. 13. No. 1, pp. 132-142.

- HIRSCH** (Rahel). Fieber und Chininwirkung im Fieber.—*Zeitschr. f. Exper. Patholog. u. Therapie*, 1913. Apr. 24. Vol. 13. No. 1, pp. 84-131.
- KINGHORN** (Allan), **YORKE** (Warrington), & **LLOYD** (Llewellyn). Final Report of the Luangwa Sleeping Sickness Commission of the British South Africa Company 1911-1912. With Appendices by A. F. Wallace and Llewellyn Lloyd.—*Ann. Trop. Med. & Parasit.*, 1913. June 10. Vol. 7. No. 2, pp. 183-302. With 12 plates.
- KLEINE** (F. K.) & **ECKARD** (B.). Ueber die Bedeutung der Speicheldrüseninfektion bei der Schlafkrankheitsfliege (*Glossina palpalis*).—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. Apr. Vol. 74. No. 1, pp. 183-187.
- & —. Zur Epidemiologie der Schlafkrankheit.—*Arch. f. Schiffa- u. Trop.-Hyg.*, 1913. May. Vol. 17. No. 10, pp. 325-328.
- KOLLE** (W.), **HARTOCH** (O.), **ROTHERMUNDT** (M.), & **SCHÜRMANN** (W.). Ueber neue Prinzipien und neue Präparate für die Therapie der Trypanosomeninfektionen.—*Deut. Med. Wochenschr.*, 1913. May 1. Vol. 39. No. 18, pp. 825-828.
- , —, —, & —. Ueber neue Prinzipien und Präparate für die Therapie der Trypanosomeninfektionen.—*Centralbl. f. Bakt.*, 1. Abt., Ref., 1913. June 14. Vol. 47. No. 14/22, pp. 166\*-173\*.
- LAGANE** (L.). Pouls lent dans la Trypanosomiasis humaine.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 269-272.
- LAMBALLE** (F. W.). Trypanosomiasis and Surra. A Preliminary Note upon the Effect of Pancreatic Enzymes upon the Trypanosome of Surra. With an Explanatory Note by J. Beard, D.Sc.—1913. June 9. 4 pp. Edinburgh: Otto Schulze & Co.
- LAVERAN** (A.). Au sujet du *Trypanosoma rhodesiense* et du *Tr. brucei*.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 340-343.
- & **FRANCHINI** (G.). *Trypanosoma talpae* chez *Palaeopsylla gracilis*.—*Compt. Rend. Soc. Biol.*, 1913. June 20. Vol. 74. No. 22, pp. 1254-1256. With 11 text-figs.
- & **MARULLAZ** (M.). Au Sujet du *Trypanosoma talpae*.—*Compt. Rend. Soc. Biol.*, 1913. May 16. Vol. 74. No. 17, pp. 1007-1008.
- & **ROUDSKY** (D.). Essais d'immunisation contre les Trypanosomes pathogènes.—*Trypanotoxines*.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 176-181.
- & **THIROUX** (A.). La Prophylaxie de la Maladie du Sommeil.—*Revue Scientifique*, 1913. May 10. Vol. 1. No. 9, pp. 577-581.
- McCOWEN**. A Note on Sleeping Sickness in Principe Island and Angola, West Coast of Africa.—*Proc. Roy. Soc. Med.*, 1913. May. Vol. 6. No. 7. [Sect. of Epidemiology and State Med.], pp. 191-194.
- MACFIE** (J. W. Scott). Trypanosomiasis of Domestic Animals in Northern Nigeria.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Vol. 7. No. 1, pp. 1-20. With 3 plates.
- . The Distribution of *Glossina* in the Ilorin Province of Northern Nigeria.—*Bull. Entomol. Res.*, 1913. May. Vol. 4. Pt. 1, pp. 1-28. With 7 plates and 1 map.
- MESNIL** (F.). A Propos du Pouvoir protecteur des Sérums des Malades du Sommeil.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 447-451.
- MITZMAIN** (M. Bruin). Collected Notes on the Insect Transmission of Surra in Carabaos.—*Philippine Agricultural Review*, 1912. Dec. Vol. 5. No. 12, pp. 670-681.
- . The Rôle of *Stomoxys calcitrans* in the Transmission of *Trypanosoma evansi*.—*Philippine Jl. Science*. Sect. B. (*Philippine Jl. Trop. Med.*), 1912. Dec. Vol. 7. No. 6, pp. 475-518.
- MORGENROTH** & **TUGENDREICH**. Zur Chemotherapie der Trypanosomeninfektion.—*Berliner Klin. Wochenschr.*, 1913. Feb. 24. Vol. 50. No. 8, pp. 367-368.

- NEWHAM (H. B.). Trypanosomiasis. London School of Tropical Medicine. 3rd Report.—*J. London School Trop. Med.*, 1913. Apr. Vol. 2. Part 2, pp. 144-146.
- NEWSTEAD (R.). A New Tsetse Fly from the Congo Free State; and the Occurrence of *Glossina austeni* in German East Africa.—*Ann. Trop. Med. & Parasit.*, 1913. June 10. Vol. 7. No. 2, pp. 331-334. With 2 figs.
- OGAWA (M.). Studien über die Trypanosomen des Frosches.—*Arch. f. Protistenkunde*, 1913. Apr. 19. Vol. 29. No. 2, pp. 248-258. With 1 plate and 3 text-figures.
- PONSSELLE (A.). Technique pour la Coloration des Trypanosomes et Trypanoplasmes de Culture.—*Compt. Rend. Soc. Biol.*, 1913. May. Vol. 74. No. 18, pp. 1072-1073.
- v. PROWAZEK (S.). Ueber reine Trypanosomenstämme.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Apr. 16. Vol. 68. No. 5/6, pp. 498-501.
- RODHAIN (J.). A propos de *Leptomonas pangoniae* et *Trypanosoma Denysi*. Note Rectificative.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 181-182.
- RONDONI (Pietro) & GORETTI (Guido). Ricerche Sperimentali sul Nagana. I Comunicazione. Su alcune proprietà biologiche della milza nella infezione sperimentale da *Trypanosoma brucei*.—*Lo Sperimentale*, 1913. Apr. 7. Vol. 67. No. 1, pp. 1-24.
- ROUBAUD (E.). Supplément à la Repartition et à la Variation Géographique des Glossines.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 347-350.
- . Evolution comparée des Trypanosomes Pathogènes chez les Glossines.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 435-441. With 3 figures.
- ROYAL SOCIETY. Reports of the Sleeping Sickness Commission of the Royal Society. No. 13.—ii+142 pp. With 7 plates. 1913. London: H.M. Stationery Office.
- SCHUBERG (A.) & Böing (W.). Ueber den Weg der Infektion bei Trypanosomen- und Spirochätenerkrankungen.—*Deut. Med. Wochenschr.*, 1913. May 8. Vol. 39. No. 19, pp. 877-879, and *Centralbl. f. Bakt.*, 1 Abt., Ref., 1913. June 14. Vol. 57. No. 14/22, pp. 228\*-230\*.
- SHIRCORE (J. O.). On Two Varieties of *Glossina morsitans* from Nyasaland.—*Bull. Entomol. Res.*, 1913. May. Vol. 4. Pt. 1, p. 89.
- STEPHENS (J. W. W.) & BLACKLOCK (B.). On the Non-identity of *Trypanosoma brucei* (Plimmer and Bradford, 1899) with the Trypanosome of the same name from the Uganda Ox.—*Ann. Trop. Med. & Parasit.*, 1913. June 10. Vol. 7. No. 2, pp. 303-308.
- & FANTHAM (H. B.). Further Measurements of *Trypanosoma rhodesiense* and *T. gambiense*.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Vol. 7. No. 1, pp. 27-40.
- TANON (L.) & DUPONT (A.). Traitement de la Trypanosomiase humaine.—*Bull. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. May 15. 3rd ser. Vol. 29. No. 16, pp. 975-988.
- TODD (John L.). Concerning the Sex and Age of Africans suffering from Trypanosomiasis.—*Ann. Trop. Med. & Parasit.*, 1913. June 10. Vol. 7. No. 2, pp. 309-319.
- TRAMONTI (E.). Alcune considerazioni sulla malattia di Carlo Chagas (Thyreoiditis parassitaria). Rivista analitica.—*Policlinico. Sez. prat.*, 1913. May 18. Vol. 20, pp. 697-701.
- UHLENUTH (Paul) & EMMERICH (Emil). Ueber das Verhalten des Kaninchenhodens bei experimenteller Trypanosomen- und Spirochäteninfektion.—*Deut. Med. Wochenschr.*, 1913. Apr. 3. Vol. 39. No. 14, pp. 642-644.

- WENYON (C.M.). Experiments on the Transmission of *Trypanosoma lewisi* by means of Fleas.—*Jl. London School of Trop. Med.*, 1913. Apr. Vol. 2. Part 2, pp. 119-123.
- & HANSCHALL (H. M.). A Further Note on *Trypanosoma rhodesiense* from Three Cases of Human Trypanosomiasis.—*Jl. London School Trop. Med.*, 1913. Apr. Vol. 2. Part 2, pp. 123-128.
- YORKE (Warrington). Sleeping Sickness and Big Game: A Proposed Experiment.—*Brit. Med. Jl.*, 1913. June 21. pp. 1315-1317.
- . The Relationship of the Big Game of Africa to the Spread of Sleeping Sickness.—*Proc. Zoolog. Soc. of London*, 1913. June. pp. 321-337.
- YOUNG (Jas. C.). A Case of Trypanosomiasis treated by Intravenous Injections of Tartar Emetic.—*University of Durham Coll. of Med. Gaz.*, 1912. Nov. 15. Vol. 13. No. 2, pp. 19-25.

## Typhus Fever.

- ANDERSON (John F.). Some Recent Work on Measles and Typhus Fever. (Proceedings of the Johns Hopkins Hospital Medical Society, 1912, Dec. 2).—*Bull. Johns Hopkins Hospital*, 1913. Apr. Vol. 24. No. 266, pp. 121-124.
- . The Problem of Typhus in the United States.—*Jl. Amer. Med. Assoc.*, 1913. June 14. Vol. 60. No. 24, pp. 1845-1846.
- ARZT (L.) & KERL (W.). Variola- und Flecktyphus-studien an den bosnischen Rückwanderern aus dem Balkan.—*Wiener Klin. Wochenschr.*, 1913. May 15. Vol. 26. No. 20, pp. 787-795.
- CARLAN (Decio). El Tifus Exantemático en Madrid.—*El Siglo Medico*, 1913. Apr. 5. Vol. 60. No. 3095, p. 209.
- MÜLLER (Paul Th.). Vorläufige Mitteilung über bakteriologische Befunde bei Flecktyphus.—*München Med. Wochenschr.*, 1913. June 24. Vol. 60. No. 25, pp. 1364-1365.

## Undulant Fever.

- SUAREZ de FIGUEROA (D. José). Cooperacion al estudio de la Fiebre de Malta. (Continuacion).—*El Siglo Médico*, 1913. May 3. Vol. 60. No. 3099, pp. 279-280, and June 14. No. 3105, pp. 371-373.
- TRIA (Pietro). La Febbre Mediterranea. (Note di Epidemiologia, Diagnostica, Profilassi).—*Riforma Medica*, 1913. Apr. 5. Vol. 29. No. 14, pp. 380-383.

## Yaws.

- BROCHARD (V.). Le Salvarsan en Lavement pour le Traitement du Pian.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 308-312.
- KERNÉIS (J.), MONFORT (F.) & HECKENROTH (F.). Quelques Remarques sur le Pian au Congo français. Pian et Ulcères phagédéniques traités par le 606.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 243-247.
- MAZZOLANI (D. A.). Le Tigne, il Pian, le Piodermatosi ed altre Affezioni cutanee curate negli indigeni della Tripolitania.—*Riforma Medica*, 1913. Apr. 12. Vol. 29. No. 15, pp. 396-400; and Apr. 19. No. 16, pp. 425-428. With 5 figures.
- REED (E. U.). Treatment of Frambesia with Salvarsan.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 220-222. With 2 figures.
- SABELLA (Pietro). Studio Parallelo fra la Siflide, la Framboesia e il Granuloma ulceroso delle Pudende, osservati nella Tripolitania (con Ricerche Istologiche e Sperimentali).—*Malaria e Malat. d. Paesi Caldi*, 1913. Mar. Vol. 4. No. 2, pp. 102-113.
- THIBAUT (E.). Formule leucocytaire dans le Pian.—*Bull. Soc. Méd. Chirurg. de l'Indochine*, 1913. May. Vol. 4. No. 5, pp. 277-281.

## Yellow Fever.

- AUGÉ.** Une Observation de Fièvre Jaune au Dahomey [Clinique d'Outre-Mer].—*Ann. d'Hyg. et de Méd. Coloniales*, 1913. Jan.-Feb.-Mar. Vol. 16. No. 1, pp. 181-189.
- FLOURENS.** Note sur la Dernière Épidémie de Fièvre Jaune au Sénégal.—*Rev. de Méd. et d'Hyg. Trop.*, 1913. Vol. 10. No. 1, pp. 31-34.
- LICEAGA (Eduardo).** Annual Report on the Condition of Yellow Fever throughout the Mexican Republic.—*Amer. Jl. Public Health*, 1913. Mar. Vol. 3. No. 3, pp. 263-264.
- MACDONALD (Angus).** Is Yellow Fever Endemic in Jamaica? A Paper read before the Jamaica Branch of the British Medical Association, December, 1912. 1913. 17 pp. Jamaica: Egbert S. Baird, 60, King Street, Kingston.
- MARQUE.** Observations de Fièvre Jaune au Sénégal [Clinique d'Outre-Mer].—*Ann. d'Hyg. et de Méd. Coloniales*, 1913. Vol. 16. No. 1, pp. 184-193.
- RICE (Thomas E.).** Evidence of the Endemicity of Yellow Fever in the Gold Coast Colony.—*Yellow Fever Bureau Bull.*, 1913. Jan. Vol. 2. No. 3, pp. 272-274.
- SEIDELIN (Harald).** The Nature and Control of Yellow Fever.—*Yellow Fever Bureau Bull.*, 1913. Jan. Vol. 2. No. 3, pp. 255-271.
- WEST AFRICA.** Report on Certain Outbreaks of Yellow Fever in 1910 and 1911.—1913. 108 pp. With 6 maps. Printed by Waterlow & Sons, London.

## Miscellaneous.

- CLIMATIC BUBO, GANGOSA, POROCEPHALIASIS, RAT BITE DISEASE, SPRUE, VERRUGA PERUANA, VOMITING SICKNESS.**
- ATKINSON (A. G.).** "Rat-bite Fever."—*Med. Chronicle*, 1913. Apr. (ser. 4). Vol. 25. No. 1. (Whole series Vol. 57. No. 343), pp. 1-28. With charts.
- CANTLIE (J.).** Collosol Argentum: Its Use in Sprue and Post-Dysenteric Conditions.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 15. Vol. 16. No. 8, pp. 123-124.
- COLE (Harold N.).** Verruga Peruana: Its Comparative Histological Study in Man and the Ape.—*Jl. of Cutaneous Diseases*, 1913. June. Vol. 31. No. 6, pp. 384-392. With 1 plate.
- GRAY (Douglas G.).** Climatic Bubo.—*China Med. Jl.*, 1913. May. Vol. 27. No. 3, pp. 180-184.
- HEWLETT (R. Tanner) & RODMAN (G. H.).** A Case of Rat-Bite Disease.—*Practitioner*, 1913. July. Vol. 91. No. 1. (No. 541), pp. 86-87.
- JAMAICA.** A Report by the Government Bacteriologist on Cases of Cerebro-Spinal Meningitis, indicating the Similarity between the Symptom-Complex of that Disease and some Cases of Vomiting Sickness.—1913. 7 pp. Jamaica: Government Printing Office, Kingston.
- KERR (W. M.).** Gangosa.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 188-200. With 10 figures.
- MAYER (M.), ROCHA-LIMA (H.), & WERNER (H.).** Untersuchungen über Verruga peruviana.—*München. Med. Wochenschr.*, 1913. Apr. 8. Vol. 60. No. 14, pp. 739-741.
- SAMBON (Louis W.).** Porocephaliasis in Man.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 1. Vol. 16. No. 7, pp. 97-100. With 8 text-figures.
- SCOTT (H. Harold).** Fulminating Cerebro-Spinal Meningitis in Jamaica.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. 31. Series T. M. Vol. 7. No. 1, pp. 165-181.

- TRIADO (A. J. J.).** Tropical or Climatic Buboes.—*Australasian Med. Gaz.*, 1913. May 10. Vol. 33. No. 19. (No. 434), pp. 442-443.
- WEGELE (C.).** Ueber die diätetische Behandlung gewisser Formen chronischer Diarrhöen speziell von "Indian Sprue."—*Mediz. Klinik.*, 1913. June. Vol. 9. No. 22, pp. 866-868.

## UNCLASSIFIED.

- BAETZ (Walter).** One Hundred Cases of Acute Arthritis among Negro Laborers on the Panama Canal.—*Jl. Amer. Med. Assoc.*, 1913. Apr. 5. Vol. 60. No. 14, pp. 1065-1068.
- BAYEUX (Raoul).** Manuel opératoire des Injections hypodermiques d'Oxygène dans les Anémies tropicales.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 276-280. With 1 fig.
- BERTARELLI (E.).** Ueber die Gegenwart von mittels Komplementablenkung in den Seris gegen Schlangengift nachweisbaren Antikörpern.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Feb. 12. Vol. 68. No. 1, pp. 67-71.
- BRAMWELL (Byrom), McDONAGH (J. E. R.), & Low (George C.).** Discussion on the Use of Salvarsan and Neo-Salvarsan in Diseases other than Syphilis.—*Proc. Roy. Soc. Med.*, 1913. Vol. 6. No. 7. (Therapeut. & Pharmacol. Sect.), pp. 131-152.
- BRAULT (J.).** Note sur une Forme d'Adénites subaiguës de l'Aïne, rencontrée en Algérie.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 201-202.
- CASTELLANI (Aldo).** A Note on Broncho-Oidiosis.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 1. Vol. 16. No. 7, pp. 102-104.
- & **CHALMERS (Albert J.).** Manual of Tropical Medicine. 2nd edit. xxxii+1747 pp. With 630 illustrations in text. 1913. London: Baillière, Tindall, & Cox.
- COOK (A. R.).** Climatic and other Factors influencing the Health of Europeans in Uganda and East Africa.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 15. Vol. 16. No. 7, pp. 120-122.
- EATON (E. M.).** A Case of Tick-Bite followed by wide-spread Transitory Muscular Paralysis.—*Australasian Med. Gaz.*, 1913. Apr. 26. Vol. 33. No. 17. [No. 432.] pp. 391-394.
- EHRlich (Paul).** Abhandlungen über Salvarsan. Vol. 3. 584 pp. With 2 plates and 49 text-figs. 1913. Munich: J. F. Lehmann's Verlag.
- FRANÇA (C.).** Un Cas de Chappa (?).—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 351-355. With 1 text-figure.
- HUMMEL (E. M.).** The Prevalence of Asthenic Disorders of the Nervous System in Warm Climates.—*Interstate Med. Jl.*, 1913. June. Vol. 20. No. 6, pp. 522-525.
- KÜLZ (L.).** Kameruner Sektionsmaterial.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Apr. Vol. 17. No. 8, pp. 273-284.
- LE ROY DES BARRES.** Rapport sur la Natalité, la Morbidité et la Mortalité à Hanoi en 1912.—*Bull. Soc. Med. Chirurg. de l'Indochine*, 1913. May. Vol. 4. No. 5, pp. 248-259.
- MARSHALL & MEERWEIN (Werner).** Ueber das leukozytäre Blutbild, einschliesslich Verschiebung der Neutrophilen, bei wilden Eingeborenen von Neuguinea.—*Folia Haematologica*, 1913. May. Vol. 15. No. 2, pp. 229-236.
- MASTERMAN (E. W. G.).** Notes on some Tropical Diseases of Palestine.—*Jl. of Hygiene*, 1913. Apr. Vol. 13. No. 1, pp. 49-62. With 1 chart.

- MAYER (Martin). Die Tuberkulose in den Tropen und bei bislang immunen Völkerschaften. (Vortrag gehalten am 20. Oktober 1911).—*Tuberkulose-Fortbildungskurse des Allgemein. Krankenhauses Hamburg-Eppendorf*. (Direktor Dr. L. Brauer) Würzburg, 1913. Vol. 2, pp. 25-28. [Verlag von Curt Kabitzsch.]
- MAYER (T. F. G.). A New Mosquito-Proof and Storm-Proof House for the Tropics.—*Ann. Trop. Med. & Parasit.*, 1913. Mar. Series T. M. Vol. 7. No. 1, pp. 41-44. With 1 plate.
- MINETT (E. P.). Diagnosis of Bacteria and Blood Parasites. Second Edition. 80 pp. London: Baillière, Tindall & Cox.
- NOCHT. Medical Impressions from a Journey in East Africa.—*Jl. of State Medicine*, 1913. May. Vol. 21. No. 5, pp. 266-273.
- OPIE (Eugene L.). Human Botryomycosis of the Liver.—*Arch. Internal Medicine*, 1913. Apr. 15. Vol. 2. No. 4, pp. 425-438. With 5 text-figs.
- PHISALIX (Marie). Propriétés vaccinantes du Venin Muqueux de la Peau des Batraciens contre lui-même et contre le Venin de la Vipère aspic.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 190-195.
- PUGH (W. S.). Report of Case of Poisoning by Sea Urchin.—*U.S. Naval Med. Bull.*, 1913. Apr. Vol. 7. No. 2, pp. 254-255.
- DE RAADT (Ol. E.). Ueber einen bisher unbekannten menschlicher Krankheitserreger.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Mar. 15. Vol. 68. Nos. 3/4, pp. 318-322. With 1 plate and 1 chart.
- RIDGWAY (J. C.). Iodine as an Antiseptic in Tropical Countries.—*Brit. Med. Jl.*, 1913. May 31. pp. 1159-1160.
- RUBINO (C.) & FARMACHIDIS (C.). Le Proprietà degli estratti acquosi di Organi nell' Emolisi da Veleno di Cobra a secondo della loro Preparazione.—*Pathologica*, 1913. May. Vol. 5. No. 108, pp. 260-264.
- SANTAMARIA (J. Martinez). Some Notes on Tropical Diseases observed in the Republic of Colombia.—*Jl. Trop. Med. & Hyg.*, 1913. Apr. 1. Vol. 16. No. 7, pp. 100-102.
- SCHERER. Ueber Skorbut in Deutsch-Südwestafrika.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Mar. Vol. 17. No. 6, pp. 191-203.
- SCORDO (F.) & RIZZUTI (G.). Considerazioni Cliniche e Ricerche Batteriologiche in una Epidemia d'Ittero Infettivo a Tripoli.—*Policlinico*, 1913. Apr. Ann. 20. Vol. 20. No. 4, pp. 145-170.
- STEVENEL (L.). Quelques Observations et Examens microbiologiques faits à Pointe à Pitre.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 356-358.
- TODD (John L.). Does a Human Tick-Borne Disease exist in British Columbia?—*Canadian Med. Assoc. Jl.*, 1912. Aug. New Series. Vol. 2. No. 8, p. 686.
- . Tick Bite in British Columbia.—*Canadian Med. Assoc. Jl.*, 1912. Dec. New Series. Vol. 2. No. 12, pp. 1118-9.
- UNITED STATES. War Department: Office of the Surgeon General. Bulletin No. 1, 1913. January. (Photomicrographs of Spirochaetae, Entamebae, Plasmodia, Trypanosomes, Leishmania, Negri Bodies and Parasitic Helminths). Washington: Government Printing Office. 46 pp. With 27 plates.
- VORTISCH-VAN VLOTEN (H.). Chinesische Splenomegalie.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. Apr. Vol. 17. No. 7, pp. 242-246.
- WHITMORE (A.). An Account of a Glanders-like Disease occurring in Rangoon.—*Jl. of Hygiene*, 1913. Apr. Vol. 13. No. 1, pp. 1-34. With 1 plate and 2 charts.
- WILLETS (David G.). General Conditions affecting the Public Health and Diseases prevalent in the Batanes Islands, P.I.—*Philippine Jl. of Science*, Sect. B, Trop. Med., 1913. Feb. Vol. 8. No. 1, pp. 49-57.

# Biting Arthropods and Ticks.

- BISHOPP (F. C.). The Stable Fly (*Stomoxys calcitrans* L.), an Important Live Stock Pest.—*Jl. Economic Entomology*, 1913. Vol. 6. No. 1, pp. 112-127.
- & KING (W. V.). Additional Notes on the Biology of the Rocky Mountain Spotted-Fever Tick.—*Jl. Econ. Entomol.*, 1913. Apr. Vol. 6. No. 2, pp. 200-211.
- BRUMPT (E.). Utilisation des Insectes auxiliaires entomophages dans la Lutte contre les Insectes pathogènes.—*Presse Méd.*, 1913. May 3. Vol. 21. No. 36, pp. 359-361.
- EYSELL (Adolf). Spinne und Stechmücke.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. June. Vol. 17. No. 12, pp. 414-415.
- FERREYROLLES (Paul). La Destruction pratique des Moustiques sous les Tropiques.—*Rev. de Méd. et d'Hyg. Trop.*, 1913. Vol. 10. No. 1, pp. 16-23.
- JACKSON (Thos.). Coconut Oil as an Insecticide. [Correspondence].—*Indian Med. Gaz.*, 1913. May. Vol. 48. No. 5, p. 203.
- LEGENDRE (J.). La Lutte contre les Moustiques au Tonkin.—*Revue d'Hygiène et de Police Sanitaire*, 1913. Mar. 20. Vol. 35. No. 3, pp. 261-265.
- . La Prophylaxie des Affections causées par les Moustiques et la Destruction de ces Insectes à l'Etat Adulte.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 205-209.
- McCONNEL (Robert E.). Some Observations on the Larva of *Auchmeromyia luteola* F.—*Bull. Entomol. Research*, 1913. May. Vol. 4. Pt. 1, pp. 29-30.
- MESNIL (F.). Maladies infectieuses et invertébrés transmetteurs.—*Bull. Inst. Pasteur*, 1913. Mar. Vol. 11. No. 5, pp. 185-196; and No. 6, pp. 233-242.
- MITZMAIN (M. Bruin). The Bionomics of *Stomoxys calcitrans* Linnaeus; a Preliminary Account.—*Philippine Jl. of Science. Sect. B. Trop. Med.*, 1913. Feb. Vol. 8. No. 1, pp. 29-48.
- NUTTALL (George H. F.). Observations on the Biology of Ixodidae. Part I.—*Parasitology*, 1913. Apr. Vol. 6. No. 1, pp. 68-118. With 2 text-figs.
- WARD (Henry B.). Arachnida.—*Reference Handbook of the Medical Sciences*. pp. 502-516.
- WURTZ (M.). Moustiques et Fosses d'Aisances.—*Rev. de Méd. et d'Hyg. Trop.*, 1913. Vol. 10. No. 1, pp. 13-15.
- Protozoology (excluding Trypanosomes and Amoebae).**
- ALEXEIEFF (A.). Introduction à la Révision de la Famille *Herpetomonadidae* (=Trypanosomidae Doflein 1911).—*Arch. f. Protistenkunde*, 1913, pp. 313-341. With 3 text-figures.
- . Systématisation de la Mitose dite "Primitive" Sur la Question du Centriole (A propos de la Division nucléaire chez *Malpighiella* sp.).—*Arch. f. Protistenkunde*, 1913. May 27. Vol. 29. No. 3, pp. 344-363. With 7 text-figures.
- BRUMPT (E.). A propos de l'*Haemocystozoon brasiliense* de Franchini.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 377-380. With 3 figures.
- CHATTON (Edouard). L'Ordre, la Succession et l'Importance Relative des Stades, dans l'Evolution des Trypanosomides chez les Insectes.—*Compt. Rend. Soc. Biol.*, 1913. June 6. Vol. 74. No. 20, pp. 1145-1147.
- & ROUBAUD (Emile). Sporogonie d'une Hémogregarine chez une Tsété (Glossina palpalis R. Desv.).—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 226-233. With 2 plates.



- DOFFLEIN (Franz) & KOEHLER (Otto).** Ueberblick über den Stamm der Protozoen.—*Handbuch der pathogenen Mikroorganismen* (Kolle u. Wassermann), 1912. 166 pp. 99 text-figures.
- FANTHAM (H. B.).** Note on the Specific Name of the *Herpetomonas* found in the Dog-Flea, *Ctenocephalus canis*.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 254-255.
- FLU (P. C.).** Over een Prowazekia-vorm (*Prowazekia Javanense*) in de ontlasting van een Patient te Weltevreden.—*Geneeskundig Tijdschr. v. Neder-Indie*, 1912. Vol. 52. No. 6, pp. 659-678. With 1 plate.
- FRANÇA (Carlos).** Les Hémogrégarines des Sauriens. Réponse au Dr. Woodcock.—*Bull. Soc. Portugaise Sci. Naturelles*, 1912. Vol. 6. No. 1, pp. 48-55.
- FRANCHINI (G.).** Un Nouveau Protozoaire parasite de l'Homme provenant du Brésil.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 156-158.
- . Nouvelle Contribution à l'Etude de *Haemocystozoon brasiliense*.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 333-336. With 5 figs.
- . Sur un Protozoaire nouveau parasite de *Anopheles maculipennis*.—*Compt. Rend. Soc. Biol.*, 1913. June 13. Vol. 74. No. 21, pp. 1196-1198. With 18 text-figures.
- & **MANTOVANI (Mario).** Di un Nuovo Parassita Protozoario trovata nel Sangue Periferico ed Epatico in un Caso di Infezione cronica mortale proveniente dal Brasile.—*Ann. Med. Navale e Coloniale*, 1913. Feb. Anno 19. Vol. 1. No. 2, pp. 125-138. With 1 coloured plate.
- LAVERAN (A.).** Présentation d'un Chien infecté de Toxoplasmose.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, p. 294.
- & **MARULLAZ (M.).** Au Sujet des Toxoplasmes du Lapin et du Gondi. —*Compt. Rend. Acad. Sci.*, 1913. Mar. 25. Vol. 156. No. 12, pp. 933-936.
- & ———. Contribution à l'Etude morphologique du *Toxoplasma gondii* et du *T. cuniculi*.—*Compt. Rend. Acad. Sci.*, 1913. 28 Apr. Vol. 156. No. 17, pp. 1298-1302.
- & ———. Infections du Lapin par le *Toxoplasma gondii*.—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 249-254.
- & ———. Recherches expérimentales sur le *Toxoplasma gondii*.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 460-468.
- & **NATTAN-LARRIER.** Au Sujet des Altérations anatomiques produites par le *Toxoplasma cuniculi*.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 158-160.
- LEGER (André).** Parasite des Hématies, Genre *Grahamella* (Brumpt), de *Mus maurus* (Gray).—*Bull. Soc. Path. Exot.*, 1913. Apr. Vol. 6. No. 4, pp. 247-249.
- MARTIN (C. H.).** Further Observations on the Intestinal Trypanoplasmas of Fishes, with a Note on the Division of *Trypanoplasma cyprine* in the Crop of a Leech.—*Quarterly Jl. of Microscop. Science*, 1913. May. Vol. 59. Pt. 1. (new ser. No. 233), pp. 175-195. With 2 plates and 2 text-figures.
- MARULLAZ (M.).** Au Sujet d'un Toxoplasme des Oiseaux.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 323-326. With 9 figs.
- MOLDOVAN (J.).** Sur le Développement du *Leucocytozoon ziemanni* (Laveran). Note préliminaire.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 428-429.
- NEGRI (Adelchi).** Beobachtungen über *Haemoproteus*.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Apr. 23. Vol. 68. No. 7, pp. 599-602. With 1 coloured plate.

- NICOLLE (Charles) & CONOR (Marthe). La Toxoplasmose du Gondi. Maladie naturelle. Maladie expérimentale.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 160-165.
- PHISALIX. Sur une Hémogrégarine du Python moulure et ses Formes de Multiplication endogène.—*Compt. Rend. Soc. Biol.*, 1913. May 23. Vol. 74. No. 18, pp. 1052-1057.
- . Sur une Hémogrégarine de la Vipère Fer de Lance et ses Formes de Multiplication endogène.—*Compt. Rend. Soc. Biol.*, 1913. June 20. Vol. 74. No. 22, pp. 1286-1288. With 11 text-figs.
- & LAVERAN (A.). Sur une Hémogrégarine nouvelle, de *Lachesis alter-natus*.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 330-333. With 12 figs.
- PONSSELLE (A.). Culture *in vitro* du *Trypanoplasma varium* Leger.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 11. Vol. 74. No. 12, pp. 685-688.
- RODHAIN (J.). A propos de *Leptomonas pangoniæ* et *Trypanosoma Denysi*.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 181-182.
- , PONS (C.), VANDENBRANDEN (F.) & BEQUAERT (J.). Notes sur quelques Hématozoaires du Congo Belge.—*Arch. f. Protistenkunde*, 1913. Apr. 19. Vol. 29. No. 2, pp. 259-278. With 1 plate and 5 text-figures.
- , —, — & —. Note sur des Trypanosomides intestinaux d'*Haematopota* au Congo Belge.—*Bull. Soc. Path. Exot.*, 1913. Mar. Vol. 6. No. 3, pp. 182-184.
- ROSDONI (Pietro). Sulla Classificazione dei Protozoi Emoparassiti: Il Nuovo Ordine dei *Binucleati* (Hartmann).—*Lo Sperimentale*, 1913. Apr. 7. Vol. 67. No. 1, pp. 105-118.
- ROUDSKY (D.). A propos de la Note de M. Alexeieff intitulée: Introduction à la Révision de la Famille des *Herpetomonadidae*.—*Arch. f. Protistenkunde*, 1913. May 27. Vol. 29. No. 3, pp. 342-343.
- SANGIORGI (Guiseppe). Un Nuovo Protozoo Parassita del *Mus musculus*.—*Pathologica*, 1913. June 1. Vol. 5. No. 110, pp. 323-325.
- SPLENDORE (A.). Des Formes Flagellées et des Gamètes dans le *Toxoplasma cuniculi*.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 318-323. With 1 plate.
- THIROUX (A.). Les Formes de Reproduction par Schizogonie et Sporogonie d'*Haemogregarina Pettiti* (Thiroux, 1910) chez *Crocodilus niloticus*.—*Bull. Soc. Path. Exot.*, 1913. May. Vol. 6. No. 5, pp. 327-330. With 10 figs.
- VIGUIER (G.) & WEBER (A.). Les Mitochondries de l'*Haemogregarina sergentium* durant son Evolution dans le Sang du Gongyle.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 11. Vol. 74. No. 12, pp. 664-666.
- & —. Nouvelles Observations sur l'Altération des Hématies sous l'Influence d'une Hémogrégarine chez le Gongyle.—*Compt. Rend. Soc. Biol.*, 1913. Apr. 18. Vol. 74. No. 13, pp. 760-761.

See also Amoebiasis and Sleeping Sickness.



## LIST OF REFERENCES.

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**Amoebiasis (including Amoebic Dysentery and Liver Abscess).**

- CAZAMIAN.** Abscès du Foie opéré, ouvert secondairement dans l'Estomac. Vaste Ulcère par Autodigestion de la Paroi. Mort.—*Arch. de Méd. et Pharm. Navales*, 1913. Aug. Vol. 100. No. 8, pp. 138-150.
- CHATTON** (Edouard). Culture de Quelques Protistes Marins. Amibes Cystigènes et Acystigènes.—*Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, pp. 178-180.
- CHAUFFARD** (A.). Abscès Dysentérique du Foie, avec Vomiques successives. Traitement par la Ponction évacuatrice et l'Emétine.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. May 22. (3 ser.) Vol. 29. No. 17, pp. 1017-1024.
- . Les Mauvais Effets de l'Emétine en Lavement dans la Dysenterie Amibienne.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. July 3. (3 ser.) Vol. 29. No. 23, pp. 1235-1237.
- CLUZET & BAUR** (J.). Hydropneumocyste Hépatique au Cours d'un grand Abscès du Foie, consécutif à une Dysenterie: Examens radio-scopiques, et radiographiques.—*Lyon Médical*, 1913. July 13. (45 Année.) Vol. 121. No. 28. pp. 50-55. With 2 plates.
- CORDIER.** Dysenterie et Hépatite suppurée.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 444-446.
- CRAIG** (Charles F.). The Identity of *Entameba histolytica* and *Entameba tetragena*, with observations upon the Morphology and Life Cycle of *Entameba histolytica*.—*Jl. of Infectious Diseases*, 1913. July. Vol. 13. No. 1, pp. 30-52. With 2 plates.
- DARLING** (S. T.). The Rectal Inoculation of Kittens as an Aid in determining the Identity of Pathogenic Entamoebae.—*Southern Med. Jl.*, 1913. August. Vol. 6. No. 8, pp. 509-511.
- DESSY** (S.). & **MAROTTA** (R. A.). Contribución al Tratamiento de la Enteritis Disentérica y del Absceso del Hígado (Amibiano), con el Método de Rogers.—*Semana Médica*, 1913. May 15. Vol. 20. No. 20, pp. 1136-1141.
- FLANDIN** (Charles) & **DUMAS** (René). Gros Abscès Dysentérique du Foie ouvert dans les Bronches. Guérison obtenue par le Traitement chirurgical et les Injections de Chlorhydrate d'Emétine.—*Rev. Méd. de S. Paulo*, 1912. Oct. 31. Vol. 15. No. 20, pp. 409-413.
- GAIDE** (L.) & **MOUZELS** (P.). Note sur le Traitement de la Dysenterie Amibienne par l'Emétine.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 491-494.
- GIFFIN** (H. Z.). Clinical Notes on Patients from the Middle North-west infected with Entamoebas.—*Jl. Amer. Med. Assoc.*, 1913. Aug. 30. Vol. 61. No. 9, pp. 675-677.
- HUTCHINSON** (Allen C.). Results in Thirteen Cases of Dysentery treated with Emetine.—*China Med. Jl.*, 1913. July. Vol. 27. No. 4, pp. 243-245.
- JAMES** (W. M.). The Clinical Identification of Entamoebae.—*Proceedings of the Canal Zone Med. Assoc.*, 1912. Vol. 4. Part 2. [12 pp.]

- LA CAVA (Francesco). La Chemioterapia della Dissenteria da Amebe.—*Pathologica*, 1918. July 15. Vol. 5. No. 118, pp. 424-428.
- LYON (Gaston). Traitement par l'Emétine de la Dysenterie Amibienne.—*Rev. Med. de S. Paulo*, 1912. Oct. 31. Vol. 15. No. 20, pp. 406-408.
- MALLANNAH (S.). Emetine and Liver Abscess. [Correspondence].—*Indian Med. Gaz.*, 1918. Aug. Vol. 48. No. 8, pp. 331-332.
- MATHIS (C.). Recherche des Kystes d'Amibes dans les Selles de l'Homme.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. July. Vol. 4. No. 7, pp. 384-350. With 4 plates.
- MAURIN (M.). Dysenterie Amibienne traitée et guérie par la Décoction d'Ipéca en Lavements.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. July 31. (3 ser.) Vol. 29. No. 27, pp. 282-285.
- MORIYASU. The Amoebic Dysentery in Korea and its Blood Changes.—*Sei-i-Kwai Med. J.*, 1913. Sept. 10. Vol. 32. No. 9. [Whole No. 379], p. 115. [The original in No. 2, Vol. 1, of the J. Japanese Med. Soc. Nippon, Naikwa-gakkwai.]
- ROUX (G.) & TRIBONDEAU (L.). Action de l'Emétine dans quelques Formes Spéciales d'Amibisme et, par Analogie avec une d'elles, dans la Syphilis.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 424-427.
- SAMBUC (E.). Les Absès du Foie à l'Hôpital de Haiphong.—*Arch. Générales de Chirurgie*, 1913. June 25. Vol. 7. No. 6, pp. 641-659.
- THOMPSON (J. H. C.). The Treatment of Dysentery by Injections of Emetine Hydrochloride.—*Dublin J. of Med. Sciences*, 1913. Aug. (3rd ser.) No. 500, pp. 102-109.
- VALENCE (M.). Absès du Foie traité selon la Méthode de Rogers. Cures d'Emétine contre l'Amibiase.—*Bulls. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. July 31. Vol. 29. (3 sér.) No. 27, pp. 276-282.
- WADHAMS (S. H.) & HILL (E. C.). Three Cases of Amebic Dysentery treated with Salvarsan.—*Jl. Amer. Med. Assoc.*, 1913. Aug. 9. Vol. 61. No. 6, pp. 385-386.
- WHITMORE (Eugene R.). Free-Living and Parasitic Amoebae and their Relation to Dysentery.—*Amer. Jl. Trop. Dis. & Preventive Med.*, 1913. Sept. Vol. 1. No. 3, pp. 197-219. With 1 plate.

### Beriberi.

- CASPARI (W.) & MOSZKOWSKI (M.). Weiteres zur Beriberifrage.—*Berlin. Klin. Wochenschr.*, 1913. Aug. 18. Vol. 50. No. 33, pp. 1515-1519.
- CHAMBERLAIN (Weston P.). The Character of the Rice which prevents Beri-Beri and the Manner in which it is milled.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Aug. Vol. 1. No. 2, pp. 121-128.
- EISENMAN (C.). Aetiologie und Prophylaxis der Beriberi.—*Trans. xvi Intern. Congress of Med. London*, 1913. Section xxi. Trop. Med. & Hyg. Part 1, pp. 25-39.
- FUNK (Casimir). Ueber die physiologische Bedeutung gewisser bisher unbekannter Nahrungsbestandteile, der Vitamine.—*Ergebnisse der Physiologie*, 1913. Vol. 13. pp. 123-205. [Nachtrag zum Aufsatz: pp. 547-548.]
- . Fortschritte der experimentellen Beriberiforschung in den Jahren 1911-1913.—*München. Med. Wochenschr.*, 1913. Sept. 9. Vol. 60. No. 36, pp. 1997-1999.
- & MACALLUM (A. B.). On the Chemical Nature of Substances from Alcoholic Extracts of Various Foodstuffs which give a Colour Reaction with Phosphotungstic and Phosphomolybdic Acids. (Preliminary Communication).—*Biochemical Jl.*, 1913. July. Vol. 7. No. 4, pp. 357-358.

- HEISER** (Victor G.). Further Experiences with Beri-Beri in the Philippine Islands.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Aug. Vol. 1. No. 2, pp. 119-120.
- JENNISSEN** (J. A. M. J.). Over rijstvoeding en Beri-Beri onder het Mijnwerkerscorps te Billiton.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 4, pp. 570-583.
- KARIYA & TSUJUKI**. The Blood Pressure of Beri-Beri Patients.—*Sei-i-Kwai Med. Jl.*, 1913. Aug. 10. Vol. 32. No. 8. [Whole No. 378], pp. 109-110. (The original in No. 10, Vol. 27, 1913, of the *Jl. Tokyo Med. Assoc.*)
- LAIDLAW** (James D.). Etiology of Beriberi. [Memoranda.]-*Brit. Med. Jl.*, 1913. July 5. pp. 20-21.
- LINDSAY** (J. W.). Etiology of Beri-Beri. [Memoranda.]-*Brit. Med. Jl.*, 1913. July 5. p. 20.
- LOVELACE** (Carl). Peripheral Neuritis in the Amazon Valley.—*Texas State Jl. of Med.*, 1913. July. Vol. 9. No. 3, pp. 94-95; and *Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Aug. Vol. 1. No. 2, pp. 140-145.
- NOCHT** (B.). Beriberi.—*Trans. xvii Intern. Congress of Med. London*, 1913. Section xxi, Trop. Med. & Hyg., Part 1, pp. 41-44.
- SCHAUMANN** (H.). Zu dem Problem der Beriberi-Aetiologie, II.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, pp. 433-445.
- WELLMAN** (Creighton). On the Production of a Beriberi-form Polyneuritis in Fowls with Substances other than Rice.—*Southern Med. Jl.*, 1913. Aug. Vol. 6. No. 8, pp. 516-518. With 9 text-figs.
- & **BASS** (C. C.). Polyneuritis Gallinarum caused by Different Food-stuffs.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Aug. Vol. 1. No. 2, pp. 129-139. With 3 plates.
- YAMAGIWA** (K.), **KOYANO** (T.), **MIDORIGAWA** (H.), & **MOGI** (T.). Experimental Study on the Cause and Nature of Beri-beri. Report II.—*Sei-i-Kwai Med. Jl.*, 1913. Aug. 10. Vol. 32. No. 8. [Whole No. 378], p. 110. (The original in No. 12, Vol. 27, 1913, of the *Jl. Tokyo Med. Assoc.*)

### Blackwater Fever.

- BOYE**. Fièvre Bilieuse Hémoglobininurique, Essai de Traitement par le Sérum Antivenimeux.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 447-449.
- LALOR** (N. P. O'Gorman). Note upon some Unusual Forms of the Parasite of Pernicious Malaria, found at an Endemic Blackwater Fever Centre in Blood Smears from Certain Children.—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, pp. 253-254. With 1 plate.
- MACGILCHRIST** (A. C.). The Haemolytic Action of Quinine and its Salts, with Suggestions regarding the Etiology and Treatment of Blackwater Fever.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 16-17. (1913. Simla: Govt. Central Branch Press.)
- . The Haemolytic Action of Quinine and its Salts, with Suggestions regarding the Etiology and Treatment of Blackwater Fever.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 119-166. With 6 text-figs.
- RICHTER** (Geo.). The Etiology of "Blackwater Fever."—*Med. Record*, 1913. Aug. 16. Vol. 84. No. 7, p. 297.

### Cholera.

- CHAZARAIN-WETZEL**. Accès Pernicieux Cholériforme.—*Bull. Soc. Méd. Chirurg. de l'Indochine*, 1913. July. Vol. 4. No. 7, pp. 324-326.
- GOÉRÉ** (J.). Le Choléra à Ferryville (Tunisie) en 1911. Etude Clinique et Bactériologique.—*Arch. de Méd. et Pharm. Navales*, 1913. July. Vol. 100. No. 7, pp. 52-60, Aug., No. 8, pp. 124-137; and Sept., No. 9, pp. 207-215.

- GRIGG (E. D. W.). An Investigation on the Occurrence of the Cholera vibrio in the Biliary Passages.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 44-58. With 3 plates.
- . An Investigation of an Epidemic of Cholera caused by a "Carrier."—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 59-64.
- . An Investigation of Cholera Convalescents and Contacts in India.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 65-89. With a map.
- . Preliminary Note on the Occurrence of the Comma bacillus in the Urine of Cases of Cholera.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 90-91.
- HAFKINE (W. M.). Protective Inoculation against Cholera. 98 pp. Illustrated. 1913. Calcutta: Thacker, Spink, & Co. London: W. Thacker & Co.
- HOROWITZ (L.). Zur Frage über Cholera-Toxine und -Antitoxine.—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie*, 1. Teil., Orig., 1913. Aug. 5. Vol. 19. No. 1, pp. 44-65.
- KABESHIMA (T.). Ueber einen Hämoglobineextrakt-Soda-Agar als Elektivnährboden für Choleravibrionen.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 4. Vol. 70. Nos. 3/4, pp. 202-208.
- KOHLER (G.). Zur Frage der Choleraübertragung durch Nahrungsmittel.—*Wien. Med. Wochenschr.*, 1913. Sept. 20. Vol. 63. No. 39, pp. 2493-2496.
- LEOPOLD (L.). Het Cholera-vaccin te Stagen.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 3, pp. 475-478.
- LOGIE (W. J.). On the Inhibition of the Cholera-Red Reaction by certain Nitrite-Destroying Organisms and on the Mutual Inhibition of *B. dysenteriae* (Flexner) and *V. cholerae* when grown together.—*Jl. of Hygiene*, 1913. July. Vol. 13. No. 2, pp. 162-167.
- PANAYOTATOU (Angélique). Survie du Vibrion Cholérique dans l'Eau du Nil.—*Rev. d'Hyg. et de Police Sanitaire*, 1913. July. Vol. 35. No. 7, pp. 779-787.
- POTTEVIN (Henri). Toxine et Antitoxine Cholériques.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 409-413.
- . Les Bases Scientifiques de la Lutte contre le Choléra. (Conférence faite à la Société Impériale de Médecine de Constantinople).—*Bull. Office Intern. d'Hyg. Publique*, 1913. June. Vol. 5. No. 6, pp. 953-968.
- . Contribution à l'Étiologie du Choléra.—*Bull. Office Intern. d'Hyg. Publique*, 1913. July. Vol. 5. No. 7, pp. 1158-1174.
- & VIOLE (H.). Sur les Vibrions et leur Toxines.—*Compt. Rend. Acad. Sciences*, 1913. June 30. Vol. 156. No. 26, pp. 2029-2031.
- & ———. Transmission du Choléra aux Singes par la Voie Gastro-intestinale.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 482-484.
- & ———. Choléra expérimental des Singes inférieurs.—*Compt. Rend. Acad. Sciences*, 1913. Aug. 4. Vol. 157. No. 5, pp. 343-345.
- PUNTONI (Vittorio). L'Azione di due Microbi dell' Aria sulle Proprietà Biologiche del Vibrione Colerigeno.—*Giorn. R. Soc. Italiana d'Igiene*, 1913. Vol. 35. No. 7, pp. 289-299.
- . I Vibrioni "Inagglutinabili." Loro Rapporti con il Vibrione Colerigeno e loro Importanza nella Etiologia e Profilassi del Colera.—*Policlinico. Sez. med.*, 1913. Sept. Vol. 20. No. 9, pp. 385-409.
- ROELFSEMA (F. H.). Enkele korte Opmerkingen over Verloop en Behandeling van de Cholera in het Militair Hospitaal te Semarang in de Jaren 1910-1912.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 3, pp. 446-448.

**SANTOLIQUIDO.** Les Administrations Sanitaires dans la Lutte contre le Choléra. (Conférence faite à la Société Impériale de Médecine de Constantinople.)—*Bull. Office Intern. d'Hyg. Publique*, 1913. June. Vol. 5. No. 6, pp. 969-979.

**SCALITZER (Max) & Löwy (Otto).** Ueber die Verwendbarkeit der Blutalkalibouillon als Anreicherungsmedium für Choleravibrionen.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 3. Vol. 69. No. 7, pp. 556-560.

## Dengue.

**BIRT (C.).** Phlebotomus Fever and Dengue.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. June. Vol. 6. No. 7, pp. 243-256.

**ROUCHÉ.** Note sur une Epidémie de Dengue a bord de la "Manche" en 1911.—*Arch. de Méd. et Pharm. Navales*, 1913. June. Vol. 99. No. 6, pp. 450-461.

## Dysentery (Bacillary and Unclassed).

### (A.) Bacillary.

**BUTLER (C. S.).** Some Carbohydrate Reactions of the Dysentery Bacillus.—*Philippine Jl. of Science. Sec. B, Trop. Med.*, 1913. April. Vol. 8. No. 2, pp. 123-131.

**EBELING (E.).** Beobachtungen über die Y-Ruhr, gelegentlich einer Epidemie beim X. Armee-korps im Sommer 1911 und bei Nachuntersuchungen in den Jahren 1912 und 1913.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. June 26. Vol. 74. No. 3, pp. 447-472. With 1 plate.

### (B.) Unclassed.

**BOURRET.** Recherches sur le Parasitisme Intestinal, la Dysenterie et la Maladie du Sommeil à Saint-Louis (Sénégal).—*Ann. d'Hyg. et de Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 283-307.

**CUNNINGHAM (J.).** A Résumé of our Present Knowledge of Dysentery.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 92-118.

**DENIER & HUET.** La Dysenterie à Saigon.—*Bull. Soc. Path. Exot.*, 1913, June. Vol. 6. No. 6, pp. 413-415.

**FAIRISE (C.) & JANNIN (L.).** Dysenterie chronique à *Lamblia*. Etude Parasitologique et Anatomo-Pathologique.—*Arch. de Méd. Experiment. et d'Anatom. Pathologique*, 1913. Sept. Vol. 25. No. 5, pp. 525-551.

**FRIEDMANN (Moritz).** Die Ruhrepidemie beim Ulanenregiment Nr. 1 in der Kaiser Franz Josefs Kaserne in Lemberg.—*Der Militärarzt.*, 1913. July 12. Vol. 47. No. 13, pp. 178-182. (Forsetzung folgt.) [Ausgegeben mit Nr. 29 der *Wien Med. Wochenschr.*, 1913.]

**HINTZE (K.).** Operieren oder Nichtoperieren bei chronischer Dysenterie.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Sept. Vol. 17. No. 17, pp. 581-592. With 4 curves.

**V. PROWAZEK (S.).** Zur Kenntnis der Balantidiosis. Zusammenfassende Darstellung.—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Aug. Beiheft 6. pp. 5-24. [pp. 371-390.] With 2 coloured plates and 9 text-figs.

**WHITMORE (E. R.).** Dysentery in the Tropics.—*New York Med. Jl.*, 1913. Aug. 9. Vol. 98. No. 6. (No. 1810.) pp. 257-260. With 3 figs.

## Fevers (Unclassed).

**CONOR (A.).** Sur quelques nouvelles Observations de Fièvre Boutonneuse.—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 116-117.

**ELLIOTT (M. S.).** A Case of Six-day Fever.—*U.S. Naval Med. Bull.*, 1913. July. Vol. 7. No. 3, pp. 412-413.



**LALOR** (N. P. O'Gorman). A Paper on the Aetiological Relationships of Seven-day Fever. A Suggestion.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 207-208. (1913. Simla: Govt. Central Branch Press.)

### Filariasis.

**AUBERT** (P.) & **HECKENBOTH** (F.). Action de Divers Médicaments sur les *Microfilaria perstans* et *diurna*.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 457-459.

**BACH** (Fritz Werner). Ueber die "Mikrofilarienkulturen" von Wellman u. Johns, nebst Bemerkungen über die Messung der Mikrofilarien.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 29. Vol. 70. Nos. 1/2, pp. 50-60.

**BREINL** (Anton). On Human Filariasis in Queensland and the Morphology of *Microfilaria bancrofti*.—*Australian Inst. of Trop. Med. Report for the Year 1911*. pp. 18-23.

**BRUNETIERRE**. La Filaire de l'Oeil (*Filaria loa*) peut-elle déterminer des Complications Cérébrales?—*Gaz. hebdomadaire des Sciences Méd. de Bordeaux*, 1913. July 27. Vol. 34. No. 30, pp. 351-354.

v. d. **HELLEN**. Arsenpräparate und Filarien.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, p. 462.

**KÜLE** (L.) & **BACH** (Fr. W.). Beiträge zur Kenntnis von *Onchocerca volvulus* Leuck., 1893.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 23. Vol. 70. Nos. 5/6, pp. 321-325. With 6 text-figs.

**MAILLE**. Deux Cas de Filariose du Sang. Etude Hématologique.—*Arch. de Méd. et Pharm. Navales*, 1913. June. Vol. 99. No. 6, pp. 462-466.

**SALM** (M. J.). Un Cas de Filariose observé dans les Iles de la Sonde.—*Caducée*, 1913. Aug. 31. Vol. 13. No. 15, p. 205. With 1 text-fig.

**ZIMMANN** (H.). Beitrag zur Lehre tropischer Gewebsentzündungen infolge von Filariainfektion.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 14, pp. 469-493.

### Elephantiasis.

**BARILLATTI** (Juan). Interesante Observación de un Caso de Elephantiasis del Escroto.—*Semana Médica*, 1913. June 26. Vol. 20. No. 26. [No. 1015], pp. 1493-1494.

**KUHN** & **GÜHNE**. Zur operativen Behandlung der Elephantiasis scroti.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, pp. 457-458.

**MATAS** (Rudolph). The Surgical Treatment of Elephantiasis and Elephantoid States dependent upon Chronic Obstruction of the Lymphatic and Venous Channels.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. July. Vol. 1. No. 1, pp. 60-84.

**OUZILLEAU**. L'Eléphantiasis et les Filarioses dans le M'Bomou (Haut Oubangui). Rôle de la *Filaria volvulus*.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 307-321: and July-Aug.-Sept., No. 3, pp. 688-709.

### Dracontiasis.

**LOP** (M.). Un Cas de Filaire ou "Ver de Guinée." Traitement Chirurgical.—*Gaz. des Hôp. Civils et Militaires*, 1913. July 8. Vol. 86. No. 77, p. 1254.

### Heat Stroke.

**PEMBERTY** (M. S.). Heat-Stroke.—*Jl. R. Army Med. Corps*, 1913. Aug. Vol. 21. No. 2, pp. 156-164.

**ROSS** (T. S.). Heat-Stroke. [Correspondence].—*Indian Med. Gaz.*, 1913. Sept. Vol. 48. No. 9, p. 372.

## Helminthiasis.

### TREMATODES.

**MACCALLUM** (G. A.). Notes on four Trematode Parasites of Marine Fishes.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Sept. 13. Vol. 70. No. 7, pp. 407-416. With 11 text-figs.

**NICOLL** (William). Trematode Parasites from Food-Fishes of the North Sea.—*Parasitology*, 1913. July. Vol. 6. No. 2, pp. 188-194. With 1 plate.

### Distomiasis.

**PÉLISSIER**. Distomatose des Voies Biliaries. Angiocholécystite suppurée.—Mort. [Clinique d'Outre-Mer.]—*Ann. d'Hyg. et Méd. Colon.*, 1913. July-Aug.-Sept. Vol. 16. No. 3, pp. 776-777.

**PERRONCITO** (E.). Traitement de la Distomatose hépatique et de l'Uncinarirose.—*Revue de Thérapeutique*, 1913. Aug. 1. Vol. 80. No. 15, pp. 505-509.

**SAMBUC** (E.). Distomatose pancréatique.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. July. Vol. 4. No. 7, pp. 331-333.

### Schistosomiasis.

**BOUR** (E. F.). Sur la Bilharziose.—*Bull. Soc. Méd. de l'Île Maurice*, 1913. Vol. 31. (2nd Ser.) No. 32, p. 22.

**EKEHORN** (G.). Ein Fall von Bilharziosis in Upsala. (Mit kystoskopischer Untersuchung).—*Folia Urologica*, 1913. Aug. Vol. 8. No. 1, pp. 1-11.

**TSUCHIYA**. Clinical, Pathological-Anatomical, Pathogenic, Prophylactic and Therapeutic Study of the Schistosomiasis Japonica.—*Sei-i Kwai Med. Jl.*, 1913. Aug. 10. Vol. 32. No. 8. [Whole No. 378], pp. 107-109. (The original in No. 10, Vol. 27. 1913, of the *Jl. Tokyo Med. Assoc.*)

### CESTODES.

#### Taeniasis (Intestinal).

**GARIN** (Ch.) & **CHANCEL** (Ev.). *L'Hymenolepis nana* (Von Siebold, 1852). Nouveau Parasite de l'Homme observé en France. (Revue générale).—*Gaz. des Hôpit. Civils et Militaires*, 1913. July 5. Vol. 86. No. 76, pp. 1237-1242.

**PÉREZ** (George V.). Treatment of Taenia by Thymol. [Correspondence].—*Brit. Med. Jl.*, 1913. July 5. p. 49.

#### Taeniasis (Somatic).

**BARDIN** (M. C.). Kystes Hydatiques des Os. (Revue générale).—*Gaz. des Hôpit. Civils et Militaires*, 1913. July 12. Vol. 86. No. 79, pp. 1283-1288; and July 19. No. 81, pp. 1307-1311.

**BUTMENT** (W.). Case of Hydatidform Degeneration of the Chorion.—*Australasian Med. Gaz.*, 1913. July 5. Vol. 34. No. 1, p. 3.

**DÉVÉ** (F.). Echinococcose secondaire embolique périphérique.—*Compt. Rend. Soc. Biol.*, 1913. July 25. Vol. 75. No. 27, pp. 100-102. With 1 text-fig.

**MERCIER** (R.). Diagnostic d'un Kyste Hydatique Lombaire par les Méthodes Biologiques.—*Bull. et Méms. Soc. Méd. des Hôpit. de Paris*, 1913. Jan. 28. (3 sér.) Vol. 29. No. 2, pp. 51-52.

**O'HARA** (Henry M.). Some Unusual Cases of Hydatid Disease.—*Australasian Med. Gaz.*, 1913. July 5. Vol. 34. No. 1, pp. 1-3.

**RAMSAY** (J.). Unusual Cases of Hydatid Disease.—*Australasian Med. Gaz.*, 1913. June 21. Vol. 33. No. 25. [No. 440], pp. 587-593.

## NEMATODES.

- BERNARD (P. Noël) & BAUCHE (J.). Influence du Mode de Pénétration, cutanée ou buccale, de *Stephanurus dentatus* sur les Localisations de ce Nématode dans l'Organisme du Porc et sur son Evolution.—*Compt. Rend. Acad. Sciences*, 1913. July 7. Vol. 157. No. 1, pp. 74-76.
- KELLY (D.). A Case in which Round Worms caused Symptoms leading to a Diagnosis of Appendicitis.—*Australasian Med. Gaz.*, 1913. June 21. Vol. 33. No. 25. [No. 440], pp. 594-595.

## Ankylostomiasis.

- DE ALMEIDA (A. Ozorio). Campanha contra a Ankylostomiasse no Estado do Rio de Janeiro.—*Revista Med. de S. Paulo*, 1913. Jan. 31. Vol. 16. No. 2, pp. 27-32.
- ARCHIBALD (R. G.). A Case of Acute Agchylostomiasis treated by an Autogenous Vaccine of a Coliform Organism.—*Jl. Trop. Med. & Hyg.*, 1913. Sept. 1. Vol. 16. No. 17, pp. 260-262.
- AUSTREGESILLO (A.). Perniciöse Anämie in Fällen von Unzinarirose.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, pp. 445-455.
- BENAROYA (Manfred). Aegyptische Chlorose und ägyptische Anaemie.—*Klinisch-Therapeutischen Wochenschr.*, 1913. Vol. 20. No. 26. [6 pp.]
- BOERNER (M. H.). The Hookworm Problem. Synopsis of the Work of the Hookworm Commission of the Texas State Board of Health.—*Texas State Jl. of Med.*, 1913. Aug. Vol. 9. No. 4, pp. 133-134.
- CONRAN (P. C.). A Report on Ankylostomiasis in the North Nyasa District.—*Jl. Trop. Med. & Hyg.*, 1913. July 1. Vol. 16. No. 13, pp. 195-198; and *Ann. Med. Rept. on Health & Sanitary Condition of the Nyasaland Protectorate for year ended 31st March*, 1913. pp. 68-72.
- DINSMORE (W. W.). Hookworm Disease a National Problem.—*Southern Med. Jl.*, 1913. Aug. Vol. 6. No. 8, pp. 498-506. With 8 illustrations.
- GIUDICE. Sur un Cas d'Appendicite à Ankylostomes.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 436-440.
- JUDKINS (O. H.). Symptoms and Diagnosis of Hookworm Disease.—*Texas State Jl. of Med.*, 1913. Aug. Vol. 9. No. 4, pp. 134-136.
- MEIRA (Rubiao) & PARANHOS (Ulysses). L'Ankylostomiasse au Brésil. (Communication faite à la Société de Pathologie Comparée, dans la Séance du 11 février 1913.) 8 pp. [Imp. J. Thevenot, Saint-Dizier (Haute-Marne.)]
- PARISOT (J.) & FAIRISE (C.). Ankylostomose et Ictère Hémolytique. Recherches anatomopathologiques.—*Arch. des Maladies du Cœur des Vaisseaux et du Sang.*, 1913. July. Vol. 6. No. 7, pp. 458-467.
- PERRONCITO (E.). Traitement de la Distomatose hépatique et de l'Uninarirose.—*Revue de Thérapeutique*, 1913. Aug. 1. Vol. 80. No. 15, pp. 505-509.
- SAUNDEY (Robert). A Case of Ankylostomiasis in Birmingham. [Correspondence].—*Lancet*, 1913. July 19. p. 174.
- STILES (Ch. Wardell) & BOATWRIGHT (H. F.). Thymol Administration. Subjective Effects in 464 Administrations in 243 Patients.—*U.S. Public Health Rep.*, 1913. July 18. Vol. 28. No. 29, pp. 1497-1513.
- VERVOORT (H.). Oleum Chenopodii Anthelmintici, een Wormmiddel tegen Ankylostomum en Ascaris.—*Geneeskundig Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 3, pp. 435-445.

- WYLER (E. J.). Some Observations on Ankylostoma infection in the Udi District of the Central Province, Southern Nigeria. — *Jl. Trop. Med. & Hyg.*, 1913. July 1. Vol. 16. No. 13, pp. 193-195; and *Southern Nigeria Ann. Med. Rept. for year ending Dec. 31st*, 1913. pp. 40-45.

### Trichinelliasis.

- BERNSTEIN (E. P.). A Case of Trichinosis with Autopsy. — *Med. Record*, 1913. June 28. Vol. 83. No. 26, pp. 1169-1170.

### Ascariasis.

- FAURÉ-FREMIET (E.). Un Albuminoïde des Spermatozoïdes de l'*Ascaris megaloccephala*. — *Compt. Rend. Soc. Biol.*, 1913. July 4. Vol. 74. No. 24, pp. 1407-1409.
- . La Segmentation de l'Oeuf d'*Ascaris* au Point de Vue énergétique. — *Compt. Rend. Soc. Biol.*, 1913. July 25. Vol. 75. No. 27, pp. 90-92.
- GALLET DE SANTERRE. Cas de Diarrhée Dysentérique d'Origine Ascarienne. — *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 452-454.
- LAMOUREUX (A.). Fréquence du Parasitisme intestinal par *Ascaris lumbricoïdes* et par *Trichocephalus trichiuris* chez les Habitants de la Grand Comore. — *Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 455-457.
- LE ROY DES BARRES. Note sur un Cas d'Ascariodose hépatique. — *Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. July. Vol. 4. No. 7, pp. 329-330.
- SEN GUPTA (B. C.). A Case of Infantile Convulsions due to "*Ascarides*." [Correspondence.] — *Indian Med. Gaz.*, 1913. Aug. Vol. 48. No. 8, p. 332.
- VERVOORT (H.). Oleum Chenopodii Anthelmintici, een Wormmiddel tegen Ankylostomum en *Ascaris*. — *Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 3, pp. 435-445.

### Serum and Tissue Reactions, Toxins, etc.

- BEDSON (S. Phillips). Lésions des Organes à Sécrétion interne dans l'Intoxication vermineuse. — *Ann. Inst. Pasteur*, 1913. Aug. 25. Vol. 27. No. 8, pp. 682-699. With 6 text-figs.
- FLURY (Ferdinand). Beiträge zur Chemie und Toxikologie der Trichinen. — *Arch. f. exper. Path. u. Pharmacol.*, 1913. Sept. 5. Vol. 73. No. 3, pp. 164-213. With 2 figs.
- & GROLL (Hermann). Stoffwechsluntersuchungen an trichinösen Tieren. — *Arch. f. exper. Path. u. Pharmacol.*, 1913. Sept. 5. Vol. 73. No. 3, pp. 214-232. With 1 curve.
- LAY (Efisio). L'Anafilassi Passiva nella Diagnosi del Cisti da Echinococco. — *Gaz. Internaz. di Med. Chirurg. Igiene*, 1913. Aug. 30. No. 35, pp. 821-823.
- RUBINSTEIN (M.) & JULIEN (A.). Examen des Sérums de Chevaux atteints d'Ascariodose par la Méthode d'Abderhalden. — *Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, pp. 180-182.
- WEINBERG (M.) & CRUCA (A.). Recherches sur l'Anaphylaxie hydatique expérimentale. (Troisième Note.) Anaphylaxie hydatique passive. — *Compt. Rend. Soc. Biol.*, 1913. June 27. Vol. 74. No. 23, pp. 1318-1320.
- & ———. Recherches sur l'Anaphylaxie hydatique expérimentale. (Quatrième note.) L'Anaphylaxie hydatique n'est pas une Anaphylaxie sérique. — *Compt. Rend. Soc. Biol.*, 1913. July 11. Vol. 75. No. 25, pp. 21-23.
- & SÉGUIN (P.). Recherches sur l'Eosinophile et l'Eosinophilie. Propriétés phagocytaires de l'Eosinophile. (3me Note.) — *Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, pp. 170-173.

## GENERAL AND UNCLASSIFIED.

- DÉVÉ (F.). Pathogénie des Affections Parasitaires non Microbiennes du Foie chez l'Homme. — *Arch. de Parasitologie*, 1913. July 10. Vol. 16. No. 2, pp. 211-281.
- HUECK (Otto). Ueber die pathologische Bedeutung von Helminthen in der Appendix. — *Frankfurter Zeitschr. f. Patholog.*, 1913. Vol. 13. No. 3, pp. 434-474.
- JOHNSTON (J. E. L.). A Note on Helminthiasis in Bassa Province, Northern Nigeria. — *Lancet*, 1913. Sept. 27, pp. 926-927.
- LEIPER (Robert T.). Observations on Certain Helminths of Man. — *Trans. Soc. Trop. Med. & Hyg.*, 1913. July. Vol. 6. No. 8, pp. 265-297. With 36 figs.
- NICOLL (William). Recent Progress in our Knowledge of Parasitic Worms. — *Parasitology*, 1913. July. Vol. 6. No. 2, pp. 141-152.
- SALICKER. Ueber Helmintheninfektionen bei den Eingeborenen der Marianen. — *Arch. f. Schiffs- u. Trop. Hyg.*, 1913. July. Vol. 17. No. 13, p. 463.
- SAUL (E.). Beziehungen der Helminthen und Acari zur Geschwulsttätologie. xvii. Mitteilung. — *Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Sept. 27. Vol. 71. No. 1, pp. 59-65. With 2 plates.
- TENNEY (Elmer S.). Some Observations on the Prevalence of Intestinal Parasites in the Philippine Islands. — *Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. July. Vol. 1. No. 1, pp. 44-48.

## Kala Azar (and Tropical Sore).

- ARATE (A.). La Resistenza dei Globuli rossi nella Leishmaniosi Infantile. — *Malaria e Mulat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4, p. 263.
- BASILE (Carlo). Su alcune Ricerche Etiologiche in un Caso di Leishmaniosi del Mediterraneo. [Lettere all'Editore.] — *Pathologica*, 1913. July 15. Vol. 5. No. 113, pp. 447-448.
- . I recenti studi sull' identità della Leishmaniosi Umana e Canina del Mediterraneo. — *Policlinico*. Sez. Pratica, 1913. July 20. Vol. 20. No. 29, pp. 1029-1032.
- BOSE (Kailas Chandra, Rai Bahadur). The Relation of Kala Azar to Malaria. — *Proceedings of the Third Meeting of the General Malaria Committee held at Madras* Nov. 18, 19, 20, 1912. pp. 267-270. (1913. Simla: Govt. Central Branch Press.)
- CANNATA (S.). Reperto del Parassita di Leishman nel Sangue periferico. Nota Preventiva. — *Pathologica*, 1913. June 15. Vol. 5. No. 111, pp. 351-352.
- . Inclusioni Leucocitarie nella Leishmaniosi Infantile. — *Pathologica*, 1913. July 15. Vol. 5. No. 113, p. 420.
- . Sul Reperto del Parassita di Leishman nel Sangue periferico. — *Riforma Med.*, 1913. Aug. 2. Vol. 29. No. 31, pp. 844-846.
- CARONIA (G.). L'Anafilassi nella Leishmaniosi Infantile. — *Pathologica*, 1913. July 15. Vol. 5. No. 113, pp. 420-423.
- . Curve Termiche nella Leishmaniosi Infantile. — *Pediatria*, 1913. July 31. Vol. 21. No. 7. (Ser. 2, Vol. 11), pp. 481-496. With 12 curves.
- . Sul Potere Complementare del Siero di Sangue nella Leishmaniosi Infantile. — *Pediatria*, 1913. Aug. 31. Vol. 21. No. 8 (Ser. 2, Vol. 11), pp. 583-578.

- CRAGG (F. W.).** An Investigation into Kala Azar.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 39-42. (1913. Simla: Govt. Central Branch Press.)
- DIONISI (A.).** Contributo alla Anatomia patologica dell' Anemia da Leishmania.—*Malaria e Malat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4, pp. 265-269.
- DONOVAN (C.).** Kala Azar, its Distribution and the Probable Modes of Infection.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 211-214. 1913. Simla: Govt. Central Branch Press: *Indian Jl. of Med. Research*, 1913. July. Vol. 1. No. 1, pp. 177-184; and *Indian Med. Record*, 1913. July. Vol. 33. No. 7, pp. 158-157.
- GABBI (U.).** Leishmaniosi Umana e Metodi di Polemica.—*Malaria e Malat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4, pp. 269-270.
- . On the Identity of Infantile and Donovan's Leishmania (Kala-Azar).—*Jl. Trop. Med. & Hyg.*, 1913. July 1. Vol. 16. No. 13, pp. 198-199.
- . Ueber den Ursprung der Leishmaniosis interna (Kala-Azar) vom Hunde.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 3. Vol. 69. No. 7, pp. 504-516.
- . Sulla Identità Clinica ed Etiologica della Leishmaniosi Umana e Canina.—*Pathologica*, 1913. Sept. 15. Vol. 5. No. 117, pp. 543-552.
- , **LOMBARDO PELLEGRINO (P.). & MONTORO (G.).** Inchiesta intorno al Kala-Azar nelle Provincie della Sicilia orientale e della Calabria inferiore. Risultati raggiunti.—*Malaria e Malat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4, pp. 239-253.
- GRAY (A. C. H.).** Leishmaniose Naturelle du Chien à Tunis.—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 102-105.
- GURKO (A. G.).** Vier Fälle von Kala-Azar.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. May 20. Vol. 74. No. 2, pp. 355-368. With 9 text-figs.
- HILL (R. A. P.).** Note on a New Sign in Kala-Azar. *The Lancet*, 1913. Aug. 9. p. 392.
- KALA AZAR COMMITTEE.** Report on the Operations of the Kala-Azar Committee. *Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 34-35. (1913. Simla: Govt. Central Branch Press.)
- KORKE (Vishnu T.).** Progress Report on "Some Observations on the Epidemiology of Kala Azar in Madras."—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 239-256. (1913. Simla: Govt. Central Branch Press.)
- LAVERAN (A.).** Le Kala-Azar Méditerranéen ou Infantile. *Trans. xvii Intern. Congress of Med. London*, 1913. Sec. xxi. Trop. Med. & Hyg., Part 1, pp. 71-107.
- MACKIE (F. P.).** Progress Report on Kala Azar.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 233-238. (1913. Simla: Govt. Central Branch Press.)
- MUIR (E.).** The Diagnosis and Treatment of Chronic Malaria and Kala-Azar. [Mirror.]—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, pp. 267-268.
- PATTON (W. S.).** Is Kala Azar in Madras of Animal Origin?—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 215-220. (1913. Simla: Govt. Central Branch Press): and *Indian Jl. of Med. Research*, 1913. July. Vol. 1. No. 1, pp. 185-195.

- PATTON (W. S.).** Further Observations on the Development of *Herpetomonas donovani* in *Cimex rotundatus* and *Cimex lectularius*.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 221-232. (1913. Simla: Govt. Central Branch Press.)
- PITTALUGA (G.).** El Kala Azar infantil (esplenomegalia parasitaria de los niños) en la Costa de Levante de España.—*Rev. Clínica de Madrid*, 1912. Oct. 1. 7 pp.
- , **DIESTRO (J. García del), & VILÁ (Manuel).** Estudios sobre el "Kala azar infantil" y la *Leishmania infantum* en España.—*Bol. del Inst. Nacional de Alfonso XIII*, 1912. Dec. pp. 17-45. With 3 plates.
- QUILICHINI.** Un Cas de Leishmaniose infantile suivi de Guérison. Formules Leucocytaires dans la Leishmaniose.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7., pp. 495-498. With 1 curve.
- SCORDO.** Ueber einige Infektionsversuche der "Anopheles" mit dem Milzsaft von Leishmaniosiskranken.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 29. Vol. 70. Nos. 1/2, pp. 36-41. With 1 coloured plate.
- , Ueber die Frage nach der Uebertragbarkeit des Kala-azar durch einige blutsaugende Insekten.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 23. Vol. 70. Nos. 5/6, pp. 307-319.
- SOCIETÀ ITALIANA fra i Cultori delle Malattie Esotiche.** Riunione Privata tenuta a Messina il 15 Giugno 1913 intorno alla Leishmaniosi Umana in Italia. Atti, Relazioni, Comunicazioni scientifiche (con tavole) per cura del Prof. Dr. G. Spagnolio, Dr. M. Signer, Segretari della Riunione, 1913. 180 pp. With 9 plates. Messina: Stab. Tipografico Guerriera.
- YOUNG (T. C. McCombie).** An Account of an Investigation of the Prevalence of Endemic Kala Azar in the Plains of Assam.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 257-265. (1913. Simla: Govt. Central Branch Press.)

#### Tropical Sore (Dermal Leishmaniasis).

- FAZZARI (G. B.).** Un Caso di Bottone d'Oriente in Antonimina (Reggio Cal.).—*Malaria e Malat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4. pp. 264-265. With 1 text-fig.
- GONDER (Richard).** Experimentelle Uebertragung von Orientbeule auf Mäuse.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. June. Vol. 17. No. 12, pp. 397-403. With 2 plates.
- MARTOGLIO (F.).** Il Bottone Orientale in Abissinia.—*Scritti Medici "In onore del Prof. Angelo Celli, nel 25 Anno di Insegnamento,"* 1912. [Turin: Unione Tip.-Editrice Torinese.]
- SANT'ANNA (Firmino).** Trabalhos experimentaes sobre um Caso de Leishmaniose de Origem Brasileira.—*Medicina Contemporanea*, 1913. Aug. 24. Vol. 31. No. 34, pp. 267-272. With 2 text-figs.
- UFFERTE (L.) & PELLIER (J.).** Sur un Cas de Bouton d'Orient (Clou de Gafsa).—*Annales de Dermatologie et de Syphiligraphie*, 1913. June. Vol. 4. No. 6, pp. 331-334. With 1 text-fig.

#### Leprosy.

- ASEN (Johannes).** Eine Leprosenordnung von Melaten bei Köln aus dem 16. Jahrhundert.—*Leprosy*, 1913. July. Vol. 14. No. 2, pp. 70-72.
- BARBÉZIEUX (G.).** Contribution à l'Etude de l'Hérédité Léprouse. Malformations congénitales observées chez Deux Consanguins issus de Parents Léproux.—*Rev. de Méd.*, 1913. Sept. 10. Vol. 33. No. 9, pp. 737-744. With 4 text-figs.
- BAYON (H.).** A Critical Review of Recent Experimental Leprosy Research.—*S. African Med. Rec.*, 1913. June 14. Vol. 11. No. 11, pp. 201-222. With 4 plates.

- DE BEURMANN & GOUGEROT.** Bacillurie et Bacillémie Hanséienne. Le Rein des Léproux. — *Lepra*, 1913. July. Vol. 14. No. 2, pp. 73-77.
- BIEHLER (R.).** Wundheilung bei Lepra. — *Deut. Zeitschr. f. Chirurgie*, 1913. Sept. Vol. 124. Nos. 1/4, pp. 47-52. With 4 text-figs.
- BLUE (Rupert).** The Public Health Aspects of Leprosy in the United States. — *Jl. Amer. Med. Assoc.*, 1913. Sept. 20. Vol. 61. No. 12, pp. 943-946.
- BOECKMANN (Eduard).** Clinical Aspect of Leprosy. — *Jl. Amer. Med. Assoc.*, 1913. Sept. 20. Vol. 61. No. 12, pp. 946-949.
- CLIPPINGDALE (S. D.).** The Leper Window. — *Lepra*, 1913. July. Vol. 14. No. 2, pp. 78-79.
- DAVIES (Thos. Sydney).** Notes on the Specific Treatment of Leprosy by Means of a Cultural Extract. — *S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12, pp. 247-248.
- DYER (Isadore).** The Dermatologic Aspects of Leprosy. — *Jl. Amer. Med. Assoc.*, 1913. Sept. 20. Vol. 61. No. 12, pp. 950-951. With Discussion, pp. 951-952.
- FRASER (Henry) & FLETCHER (William).** The Bacillus Leprae: Has it been Cultivated? — *Lancet*, 1913. Sept. 27. pp. 918-921.
- GOMES (E.) & TERRA (F.).** Prophylaxie da Lepra. — *Brazil Medico*, 1913. Vol. 27. No. 28, pp. 288-289.
- GOODHUE (E. S.).** The Surgical Cure of Leprosy, based on a New Theory of Infection. — *New York Med. Jl.*, 1913. Aug. 9. Vol. 98. No. 6. (No. 1810), pp. 266-268. With 3 figs.
- HARRIS (Wm. H.) & LANFORD (John A.).** The Complement Fixation Test (Gay's Modification of the Besredka Method) in the Differentiation of Acidfast Bacilli. — *Jl. of Infectious Diseases*, 1913. Sept. Vol. 13. No. 2, pp. 301-308.
- HEISER (Victor G.).** Leprosy. A Note regarding the Apparent Cure of Two Lepers in Manila. — *U.S. Public Health Rep.*, 1913. Sept. 5. Vol. 28. No. 36, pp. 1855-1856.
- HEYMANS (A.).** Surgical Treatment of some Eye Affections in Lepers. — *S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12. pp. 246-247.
- IMPET (S. P.).** Symptomatology and Diagnosis of Leprosy. — *S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12, pp. 239-245.
- KEUSSEN (Herm.).** Beiträge zur Geschichte der Kölner Lepra-Untersuchungen. — *Lepra*, 1913. July. Vol. 14. No. 2, pp. 80-112.
- LAGANE (L.) & COLOMBIER (P.).** Formule sanguine de Léproux séjournant en France. — *Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 418-423.
- LEBOEUF (A.) & SALOMON.** Note sur la Lèpre des Rats en Nouvelle-Calédonie. — *Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 484-485.
- MARCHOUX.** La Lèpre. — *Rev. d'Hyg. et de Police Sanitaire*, 1913. Aug. 20. Vol. 35. No. 8, pp. 883-939.
- MEYER (Carlos).** Instituto Bacteriologico do Estado de S. Paulo. Relatorio sobre a administração e os Trabalhos do Instituto durante o anno de 1912. — *Rev. Med. de S. Paulo*, 1913. Feb. 15. Vol. 16. No. 3, pp. 56-61. [Leprosy of Rats, p. 60.]
- MILLER (Thomas).** Early Diagnosis of a Case of Leprosy much Assisted by the X Rays. — *Lancet*, 1913. July 26. p. 219. With 2 text-figs.
- MORROW (R.).** The Care of the Leper. — *S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12, pp. 234-239.
- PRINGAULT (A.).** Contribution à l'Etude hématologique de la Lèpre. (Réunion Biologique de Marseille) — *Compt. Rend. Soc. Biol.*, 1912. Nov. 30. Vol. 73. pp. 586-587.



- REHNSTIERNA (John).** Ueber die Kultivierbarkeit und Morphologie des Lepraerregers und die Uebertragung der Lepra auf Affen.—*Arch. f. Dermatol. u. Syphilis*, Orig., 1913. July. Vol. 116. No. 3, pp. 480-554. With 15 plates.
- RIDLON (J. R.).** Note on Leprosy in Rats.—*U.S. Public Health Rep.*, 1913. July 11. Vol. 28. No. 28, pp. 1447-1448.
- ROST (E. R.).** On the Leprosy Bacillus and Allied Bacilli.—*Med. Press*, 1913. Sept. 24. Vol. 147. (New Ser. Vol. 98). No. 3881, pp. 349-351.
- SANDES (T. Lindsay).** The Surgery of Leprosy.—*S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12, pp. 229-233.
- SERRA (Alberto).** L'Antiléprol dans le Traitement de la Lèpre.—*Lepre*, 1913. July. Vol. 14. No. 2, pp. 63-69. With 8 figs.
- SUGAI & MONONOBE.** The Examination of Lepra Bacillus in Circulating Blood of the New Borns.—*Sei-i-Kwai Med. Jl.*, 1913. July 10. Vol. 32. No. 7. [Whole No. 377], pp. 102-103. [Original in *Jl. Tokyo Med. Assoc.*, 1913. Vol. 27. No. 8.]
- THIRIAULT (E.).** De Quelques Recherches sur la Valeur comparée du Mucus nasal, du Suc ganglionnaire, et du Sang pour le Diagnostic de la Lèpre.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. June. Vol. 4. No. 6, pp. 293-298.
- TSURUMI (M.).** Ueber die Präzipitation und Komplementbindung mit Cuorin bei Lepra und die Beziehungen von Cuorin und Lecithin zu Lepraseren bei den Reaktionen.—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie*, 1. Teil., Orig., 1913. Aug. 5. Vol. 19. No. 1, pp. 19-30.
- VEILLON (A.) & LAGANE (L.).** Action défavorable de l'Arséno-Benzol dans la Lèpre.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 415-417.
- WOOD (D. J.).** The Eye Complications of Leprosy.—*S. African Med. Rec.*, 1913. June 28. Vol. 11. No. 12, pp. 245-246.

## Malaria.

- ACTON (H. W.) & KNOWLES (R.).** Latent Malaria.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 22-23. (1913. Simla: Govt. Central Branch Press.)
- & —. The Diagnosis of Latent Malaria.—*Indian Jl. Med. Research*, 1913. July. Vol. 1. No. 1, pp. 167-176. With 9 charts.
- ADIE (J. R. & Mrs.).** Note of an Inquiry into Malaria and Mosquitoes in the Kashmir Valley.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 91-94. (1913. Simla: Govt. Central Branch Press): and *Indian Med. Gaz.*, 1913. Sept. Vol. 48. No. 9, pp. 341-342. With 1 map.
- ATKINSON (Thomas G.).** Malaria and Afterwards.—*Indian Med. Rec.*, 1913. Aug. Vol. 33. No. 8, pp. 177-178.
- AUDIAU.** Paludisme à Forme Hépatique.—*Ann. d'Hyg. et Méd. Colon.*, 1913. Apr.-May-June. Vol. 16. No. 2, pp. 454-455.
- BALFOUR (Andrew).** A Year's Anti-malarial Work at Khartoum. — *Jl. Trop. Med. & Hyg.*, 1913. Aug. 1. Vol. 16. No. 15, pp. 225-232. With 5 text-figs. and 1 map.
- BASS (Charles C.) & JOHNS (Foster M.).** Cultivation of Malarial Plasmodia (*Plasmodium falciparum*) in vitro in the Blood of a Diabetic without the Addition of Dextrose.—*Amer. Jl. Trop. Dis. & Preventive Med.*, 1913. Sept. Vol. 1. No. 3, pp. 246-249.
- BATES (John Pelham).** A Review of a Clinical Study of Malarial Fever in Panama.—*Jl. Trop. Med. & Hyg.*, 1913. July 15. Vol. 16. No. 14, pp. 209-213: and Aug. 15. No. 16, pp. 241-245.

- BENTLEY (C. A.).** A New Conception regarding Malaria.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 61-70. (1913. Simla: Govt. Central Branch Press): and *Indian Med. Record*, 1913. July. Vol. 33. No. 7, pp. 149-156.
- . Some Problems presented by Malaria in Bengal.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 71-84. (1913. Simla: Govt. Central Branch Press.)
- . Quinine Propaganda.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 99-104. (1913. Simla: Govt. Central Branch Press.)
- BIREAUD.** Idiosyncrasie Quinique caractérisée par des Hémorragies Multiples et Variées.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, p. 454.
- BOSE (Kailas Chandra, Rai Bahadur).** On Some Points in Relation to the Breeding of Mosquitoes.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 89-90. (1913. Simla: Govt. Central Branch Press): and *Indian Med. Record*, 1913. Vol. 33. No. 7, pp. 156-157.
- . The Relation of Kala Azar to Malaria.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 267-270. (1913. Simla: Govt. Central Branch Press.)
- BRITTON (W. E.).** Mosquito Control Work in Connecticut in 1912. [With Discussion.].—*Jl. Economic Entomology*, 1913. Feb. Vol. 6. No. 1, pp. 89-93.
- BROWN (Wade H.).** Malarial Pigment (Hematin) as an Active Factor in the Production of the Blood Picture of Malaria.—*Jl. Experimental Med.*, 1913. July 1. Vol. 18. No. 1, pp. 96-106.
- . The Renal Complications of Hematin Intoxication and their Relation to Malaria.—*Arch. Internal Med.*, 1913. Sept. 15. Vol. 12. No. 3, pp. 315-321.
- BRÜNN (W.) & GOLDBERG (L.).** Die Malaria Jerusalemis und ihre Bekämpfung.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. Aug. 27. Vol. 75. No. 2, pp. 209-235. With 25 text-figs.
- BUTTRICK (P. L.).** The Effect of Tides and Rainfall on the Breeding of Salt Marsh Mosquitoes.—*Jl. Economic Entomology*, 1913. Aug. Vol. 6. No. 4, pp. 352-359.
- CACACE (Ernesto).** Profilassi Antimalarica Scolastica.—*Scritti Medici in "Onore del Professore Angelo Celli, nel 25 Anno di Insegnamento,"* 1912. pp. 827-830. [Turin: Unione Tip.-Editrice Torinese.]
- CELLI (A.).** Malaria in Italy during 1910. (Translated from the Original by Major N. P. O'Gorman Lalor. With a Preface by the Translator.)—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 113-150. (1913. Simla: Govt. Central Branch Press.)
- . Die Malariaabnahme in Italien.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. July 17. Vol. 75. No. 1, pp. 123-146.
- DAGORN.** Cas d'Hémoptyisie Paludéenne.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 451-452.
- DAVIDSON (Wilson T.).** A Case of Spontaneous Rupture of the Malarial Spleen: Splenectomy.—*Texas State Jl. of Med.*, 1913. Sept. Vol. 9. No. 5, pp. 151-152. With 1 text-fig.
- DEGORGE (A.).** Accès Palustres Mortels ou Tenaces observés après la Splénectomie.—*Bull. Soc. Méd.-Chirurg. de l'Indochine*, 1913. June. Vol. 4. No. 6, pp. 299-306.
- ENGELAND (O.).** Meine Erfahrungen bezüglich der Malariaphylaxe an Bord eines Kriegsschiffes.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Aug. Vol. 17. No. 15, pp. 523-531.

- FACCIOLO (L.). Sui Uocchi Capsulati esistenti nel Sangue dei Malarici.—*N. Morgagni*, 1913. June. Vol. 55. (Parte 1. Archivo). No. 6, pp. 201-214. With 1 plate.
- FALCIONI (Domenico). Sulla Localizzazione della Malaria alle Abitazioni.—*Policlinico*. Sez. pratica, 1913. Aug. 3. Vol. 20. No. 31, pp. 1119-1122.
- FREEMAN (James V.). The Incidence of Malaria in the Puerperium.—*Southern Med. Jl.*, 1913. July 1. Vol. 6. No. 7, pp. 429-430.
- FRÓES (João A. G.). The Rapid Diagnosis of Malaria.—*Jl. Trop. Med. & Hyg.*, 1913. Sept. 1. Vol. 16. No. 17, p. 272.
- FRY (A. B.). Malaria in Bengal.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 9-10. (1913. Simla: Govt. Central Branch Press.)
- GENERAL MALARIA COMMITTEE.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. iv+289 pp. F'cap. (1913. Simla: Government Central Branch Press.)
- GIEMSA (G.). Das Muckensprayverfahren im Dienste der Bekämpfung der Malaria und anderer durch Stechmücken übertragbarer Krankheiten.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, p. 456.
- GOBERT (E.). Quelques Aspects du Problème Anti-paludique en Tunisie (1912).—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 121-128.
- GRAHAM (J. D.). Notes on Anopheline Distribution in the United Provinces.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 85-88. (1913. Simla: Govt. Central Branch Press.)
- . School Quinisation Experiments in the United Provinces.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 105-111. (1913. Simla: Govt. Central Branch Press.)
- . Progress of Present Anti-Malarial Schemes in the United Provinces.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 177-182. With 3 maps. (1913. Simla: Govt. Central Branch Press.)
- v. d. HELLEN. Notizen über Malaria bei Eingeborenen in Togo (Westafrika).—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, p. 461.
- HENSON (Graham E.). The Diagnosis of Malaria.—*Southern Med. Jl.*, 1913. July 1. Vol. 6. No. 7, pp. 424-426.
- HODGSON (E. C.). Report on the Work of the Central Malaria Bureau, 1911-12.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 51-52. (1913. Simla: Govt. Central Branch Press.)
- . Malaria Survey of Imperial Delhi.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. p. 11. (1913. Simla: Govt. Central Branch Press.)
- HORNE (J. H.). Malaria in the Madras Presidency. Notes on the Statistics of the Past 10 years.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 53-57. (1913. Simla: Govt. Central Branch Press.)
- HUDLESTON (W. E.). An Analysis of our Present Position with Regard to the Prevention and Cure of Malarial Infections.—*Jl. R. Army Med. Corps*, 1913. Sept. Vol. 21. No. 3, pp. 320-338.
- JUSTI (Karl). Zur Methodik der Chinidarreichung bei Malaria.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Aug. Vol. 17. No. 15, pp. 505-522.
- KAHN (Ida). Some Experiences with the Subtertian Fever in Kiangsi.—*China Med. Jl.*, 1913. July. Vol. 27. No. 4, pp. 231-236.

- KNAB (Frederick). The Species of Anopheles that transmit Human Malaria.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. July. Vol. 1. No. 1, pp. 33-43.
- . Anopheles and Malaria.—*Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Sept. Vol. 1. No. 3, p. 227.
- KOPYLOW (N. W.). Ueber Splenektomie bei Malaria-affection der Milz.—*Arch. f. Klinische Chirurgie*, 1913. June 17. Vol. 101. No. 3, pp. 708-734. With 6 text-figs.
- LALOR (N. P. O'Gorman). Note upon some Unusual Forms of the Parasite of Pernicious Malaria, found at an Endemic Blackwater Fever Centre in Blood Smears from Certain Children.—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, pp. 253-254. With 1 plate.
- LEGENDRE (J.). Prophylaxie du Paludisme en Italie.—*Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 468-476.
- MAC GILCHRIST (A. C.). Intravenous Injections and Subcutaneous Infusions of Quinine Salt Solutions.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* p. 97. (1913. Simla: Govt. Central Branch Press.)
- MARCHIAFAVA (E.). Ueber Malaria pernicioosa.—*Deut. Med. Wochenschr.*, 1913. Aug. 14. Vol. 39. No. 33, pp. 1577-1581.
- DA MATTA (Alfredo A.). Pneumo-paludismo Asthmaticiforme.—*Revista Med. de S. Paulo*, 1912. Oct. 31. Vol. 15. No. 20. p. 397.
- . Anosmia Palustre.—*Revista Med. de S. Paulo*, 1912. Dec. 31. Vol. 15. No. 24, p. 483.
- MUIR (E.). The Diagnosis and Treatment of Chronic Malaria and Kala-Azar.—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, pp. 267-268.
- O'CONNELL (Matthew D.). The Meteorology of Malaria.—*Jl. Trop. Med. & Hyg.*, 1913. Sept. 1. Vol. 16. No. 17, pp. 257-260.
- PRALL (Percy A.). An Inquiry into the Effects of Malaria upon the Health of our Mine Native Labourers.—*Med. Jl. of S. Africa*, 1913. Aug. Vol. 9. No. 1, pp. 6-9.
- PERRY (E. L.). Malaria in the Jeypore Agency Estate.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 11-13. (1913. Simla: Govt. Central Branch Press.)
- RIEUX (J.) & HORNUS (P.). Notes sur le Paludisme dans le Maroc Occidental.—*Arch. de Méd. et de Pharmacie Militaires*, 1913. July. Vol. 62. No. 7, pp. 1-31.
- DA ROCHA-LIMA (H.) & WERNER (H.). Ueber die Züchtung von Malaria-parasiten nach der Methode von Bass.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Aug. Vol. 17. No. 16, pp. 541-551.
- RUBINO (Gaetano). Un Caso di Perniciosa Malarica a Sindrome Meningitica con Linfocitosi del Liquido Cerebrospinale.—*Revista Ospedaliera*, 1913. July 31. Vol. 3. No. 14, pp. 610-611.
- SARKAR (Sarosi Lal). Investigations into the Incidence of Malaria in the Town of Arambagh, Hooghly District.—*Indian Med. Gaz.*, 1913. Sept. Vol. 48. No. 9, pp. 342-346.
- SATTARANJAN SEN. Some Observations on the Aetiology of the Malaria in Bengal.—*Indian Med. Gaz.*, 1913. Aug. Vol. 48. No. 8, pp. 303-305.
- SHAVER (P. J.). Eradication of Malaria.—*Texas State Jl. of Med.*, 1913. Sept. Vol. 9. No. 5, pp. 161-163.
- STANTON (A. T.). The Anopheles of Malaya. Part I.—*Bull. Entomol. Research*, 1913. Sept. Vol. 4. Pt. 2. pp. 129-133. With 4 text-figs.
- STOKES (T. G. N.). Results of the Malaria Investigation in the Central Provinces.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 59-60. (1913. Simla: Govt. Central Branch Press.)

- STRICKLAND (C.). Short Key to the Identification of the Anopheline Mosquitoes of Malaya for the Use of Medical Officers and others.—15 pp. (6 plates.) (1918. Kuala Lumpur: F.M.S. Government Printing Office.)
- . The *Myzorrhynchus* Group of Anopheline Mosquitoes in Malaya.—*Bull. Entomol. Research*, 1913. Sept. Vol. 4. Pt. 2. pp. 135-142.
- SWELLENGREBEL (N. H.). Schizogonie der weiblichen Gametocyten von *Laverania malariae* (Tropica-Parasit).—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 4. Vol. 70. No. 3/4, pp. 179-181. With 1 coloured plate.
- TURNER (J. A.). Malarial Operations in the City of Bombay.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 151-176. (1913. Simla: Central Branch Press.)
- WATERS (E. E.). The Pure Amorphous Alkaloid (of Cinchona). [Correspondence].—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, p. 285.
- WHITE (J. H.). Malaria in Louisiana.—*New Orleans Med. & Surg. Jl.*, 1913. Aug. Vol. 66. No. 2, pp. 106-107.
- WILSON (H. C.). Some Notes on Larvioides and Natural Enemies of Mosquitoes in Southern India.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 183-186. (1913. Simla: Govt. Central Branch Press.)
- WOLDERT (Albert). Malaria and how to Prevent it.—*Texas State Jl. of Med.*, 1913. Sept. Vol. 9. No. 5, pp. 163-164.
- WOOLLEY (J. M.). Malaria in the Andamans: Fever with Jaundice Cases.—*Indian Med. Gaz.*, 1913. July. Vol. 48. No. 7, pp. 266-267.
- ZOIA (L.). Studi sulla Malaria. Sul Potere Emolitico dello Siero e dei Globuli Rossi e Sulla Prova di Bordet e Gengou nella Malaria.—*Malaria e Malat. d. Paesi Caldi*, 1913. June-July. Vol. 4. No. 4, pp. 233-239.
- ZSCHUCKE (Hans). Ueber den Ausfall der Müller-Brendel'schen Modifikation der Wassermann'sche Reaktion bei Malaria.—*Berlin. Klin. Wochenschr.*, 1913. Sept 15. Vol. 50. No. 37, pp. 1716-1719.

### Myiasis.

- NEIVA (Arthur) & GOMES DE FARIA. Notas sobre um Caso de Miiase humana ocasionada par Larvas de *Sarcophaga pyophila* n. sp.—Myiasis humana, verursacht durch Larven von *Sarcophaga pyophila* n. sp.—*Mem. Inst. Oswaldo Cruz.*, 1913. Vol. 5. No. 1, pp. 16-22.
- RUDELL (Gustave L.). Creeping Eruption. Two Cases with Recovery of the Larvae.—*Jl. Amer. Med. Assoc.*, 1913. July 26. Vol. 61. No. 4, p. 247. With 3 figs.
- SERGEANT (Edm. & Et.). La "Tamné," Myiase humaine des Montagnes Sahariennes Touareg, identique à la "Thimni" des Kabyles, due à "*Oestrus ovis*."—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 487-488.

### Pappataci Fever.

- BRILLIANT (P.). Etude sur la Fièvre des Phlébotomes.—*Arch. de Méd. et Pharm. Navales.*, 1913. July. Vol. 100. No. 7, pp. 5-39. With 3 text-figs.
- BIRT (C.). Phlebotomus Fever and Dengue.—*Trans. Soc. Trop. Med. & Hyg.*, 1913. June. Vol. 6. No. 7, pp. 243-256.

**HOWLETT (F. M.).** The Breeding-Places of *Phlebotomus*.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 209-210. (1913. Simla: Govt. Central Branch Press.)

**LOUGHNAN (W. F. M.).** *Phlebotomus* in Aden.—*Jl. R. Army Med. Corps*, 1913. July. Vol. 21. No. 1, p. 92.

## Pellagra.

**ALESSANDRINI (Guilio), GIANNELLI (A.) & FILENI (E.).** La Pellagra nella provincia di Roma.—*Policlinico*, sez. prat., 1913. Aug. 24. Vol. 20. No. 34, pp. 1213-1221.

**BARDIN (James C.).** Further Observations on the Blood of Pellagra.—*Amer. Jl. of Insanity*, 1913. July. Vol. 70. No. 1, pp. 155-159.

——. Note on the Differential Blood Counts in Three Cases of Pellagra.—*Old Dominion Jl. of Med. & Surgery*, 1913. July. Vol. 17. No. 1. [3 pp.]

**BEALL (K. H.).** Pellagra.—*Texas State Jl. of Med.*, 1913. Aug. Vol. 9. No. 4, pp. 129-131.

**BLANDY (Gurth S.).** A Contribution to the Study of Pellagra in England.—*Lancet*, 1913. Sept. 6. pp. 713-717. With 2 plates.

**Box (Charles R.).** Fatal Pellagra in two English Boys.—*Brit. Med. Jl.*, 1913. July 5. pp. 2-4. With 1 coloured plate.

**CANTABUTTI (G. B.) & Others.** La Vigilanza sul Mais.—*Riv. Pellagologica Italiana*, 1913. Sept. Vol. 13. No. 5, pp. 65-66.

**CASSAMALLI (Ferdinando).** Sulla Persistenza del Potere vitale di Spore Eumicetiche, Esposte ad alta Temperatura.—*Riv. Pellagologica Italiana*, 1913. July. Vol. 13. No. 4, pp. 51-54.

**CENTANNI (E.) & GALASSI (C.).** Sul doppio Effetto, Tossico e Unilaterale, dell' Alimentazione Maidica.—*Sperimentale*, 1913. Sept. 5. Vol. 67. Suppl. to No. 4, pp. 142-150. [Atti dell' VIII Riunione d. Soc. Italiana di Patologia tenuta in Pisa nei giorni 25, 26, 27 Marzo, 1913.]

**COLE (J. W. E.).** Notes of a Case of Pellagra.—*Lancet*, 1913. Sept. 6. pp. 717-718.

**COMMISSIONE PELLAGROLOGICA PROVINCIALE DI BELLUNO.** Relazione del Presidente Dottor Luigi Alpago-Novello a S. E. il Ministro di Agricoltura Industria e Commercio.—*Riv. Pellagologica Italiana*, 1913. Sept. Vol. 13. No. 5, pp. 67-69.

**DEEKS (W. E.).** Pellagra in the Canal Zone; its Etiology and Treatment.—*Southern Med. Jl.*, 1913. July 1. Vol. 6. No. 7, pp. 438-446.

**FINATO (L.) & NOVELLO (F.).** Ricerche sulla Ipersensibilità dei Pellagrosi.—*Pathologica*, 1913. Aug. 15. No. 115, pp. 492-493.

**HAMMOND (J. A. B.).** A Case of Pellagra in England, probably contracted in Scotland.—*Brit. Med. Jl.*, 1913. July 5. p. 12.

**HUNTER (S. J.).** Pellagra and the Sand-Fly, II. [With Discussion].—*Jl. Economic Entomology*, 1913. Feb. Vol. 6. No. 1, pp. 96-101.

**JENNINGS (Allan H.) & KING (W. V.).** An Intensive Study of Insects as a Possible Etiologic Factor in Pellagra.—*Amer. Jl. of the Med. Sciences*, 1913. Sept. Vol. 146. No. 3 (No. 498), pp. 411-440.

**KELLY (D. W.).** A Case of Pellagra Treated with Salvarsan.—*New Orleans Med. & Surg. Jl.*, 1913. Aug. Vol. 66. No. 2, pp. 106-107.

**LAVINDER (C. H.).** Pellagra. Prevalence and Geographic Distribution in Arkansas, Oklahoma and Texas.—*U.S. Public Health Repts.*, 1913. July 25. Vol. 28. No. 30, pp. 1555-1558.

- LAW (William Lamar). Treatment of Pellagra with Lactic Acid Bacilli.—*Jl. Amer. Med. Assoc.*, 1913. July 5. Vol. 61. No. 1, p. 27.
- LEMPRIERE (L. R.). Pellagra. [Memoranda].—*Brit. Med. Jl.*, 1913. Sept. 27. p. 810.
- MOTT (F. W.). The Histological Changes in the Nervous System of Dr. Box's Case of Pellagra, Compared with Changes found in a Case of Pellagra dying in the Abbassieh Asylum, Cairo.—*Brit. Med. Jl.*, 1913. July 5. pp. 4-5. With 3 plates.
- NICHOLLS (Lucius). The Pathological Changes in Pellagra and the Production of the Disease in Lower Animals.—*Jl. of Hygiene*, 1913. July. Vol. 13. No. 2, pp. 149-161. With 2 plates.
- NILES (George M.). The Role of Hydrotherapy in the Treatment of Pellagra.—*Amer. Jl. of the Med. Sciences*, 1913. Aug. Vol. 146. No. 2 (No. 497). pp. 230-233.
- PERRONCITO (Aldo). Relazione sul tema: Eziologia della Pellagra.—*Sperimentale*, 1913. Sept. 5. Vol. 67. Suppl. to No. 4, pp. 94-139. [With Discussion, pp. 139-142.] [Atti dell' VIII Riunione della Soc. Italiana di Patologia tenuta in Pisa nei giorni 25, 26, 27 Marzo, 1913.]
- REID (Robert) & CALWELL (William). Notes of a Supposed Case of Pellagra.—*Brit. Med. Jl.*, 1913. Sept. 27. pp. 784-785.
- RIVISTA PELLAGROLOGICA ITALIANA. 1913. Sept. Vol. 13. No. 5, pp. 74-79. Per l'Applicazione della Legge 21 Luglio, 1902, contro la Pellagra.
- ROBERTS (Stewart R.). The Analogies of Pellagra and the Mosquito.—*Amer. Jl. of the Med. Sciences*, 1913. Aug. Vol. 146. No. 2 (No. 497), pp. 233-238.
- SAMBON (Louis W.). The Natural History of Pellagra. With an Account of Two New Cases in England.—*Brit. Med. Jl.*, 1913. July 5. pp. 5-12. With illustrations.
- . Pellagra in Great Britain: Three new Indigenous Cases.—*Brit. Med. Jl.*, 1913. July 19. pp. 119-120. With illustration.
- . Pellagra in Great Britain. Notes of some Further Cases.—*Brit. Med. Jl.*, 1913. Aug. 9. pp. 297-298.
- SHOEMAKER (Harlan). Pellagra, Surgery, The Colloids and Strong Drugs. Also Introducing a Possible New Etiological Factor.—*New York Med. Jl.*, 1913. Aug. 2. Vol. 98. No. 5, pp. 214-219.
- SHERPESHIRE (Walter). Pellagra as a Public Health Problem.—*Texas State Jl. of Med.*, 1913. Aug. Vol. 9. No. 4, pp. 131-133.
- SILER (Joseph F.) & GARRISON (Philip E.). An Intensive Study of the Epidemiology of Pellagra. Report of Progress.—*Amer. Jl. of the Med. Sciences*, 1913. July. Vol. 146. No. 1 (No. 496). pp. 42-66; and Aug. No. 2 (No. 497). pp. 238-277.
- SOZZI (Luigi). La Cura della Pellagra col Siero Nicolaïdi.—*Riv. Pellagologica Italiana*, 1913. Sept. Vol. 13. No. 5, pp. 72-74.
- STANNUS (Hugh S.). Pellagra in Nyasaland.—*Annual Medical Report on the Health and Sanitary Condition of the Nyasaland Protectorate for the year ended 31st March*, 1913. pp. 78-86. London: Printed by Waterlow & Sons, Ltd.
- THOMAS (W. Rees). Pellagra and Drug Intoxication. [Correspondence].—*Lancet*, 1913. Sept. 13. p. 842.
- TIZZONI (Guido) & DE ANGELIS (Giovanni). Ueber den Entwicklungssyklus des pleomorphen Streptobacillus der Pellagra.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 29. Vol. 70. Nos. 1/2, pp. 24-29.
- VOLPI-GERARDINI (Gino) & ZUCCARI (Giuseppe). Sulla Ipersensibilità delle Cavie ad Alimentazione Maidica per il Siero di Sangue di Pellagroso.—*Riv. Pellagologica Italiana*, 1913. Sept. Vol. 13. No. 5, pp. 69-71.

## Plague.

- CREECH** (R. H.). The Rat. A Sanitary Menace and an Economic Burden.—*U.S. Public Health Rep.*, 1913. July 4. Vol. 28. No. 27, pp. 1403-1408.
- FOX** (Carroll). The Plague Outbreak in Iloilo.—*Philippine Jl. of Science*, Section B. Trop. Med., 1913. April. Vol. 8. No. 2, pp. 119-120. With 1 map.
- FOY** (F. Arthur). Destruction of Rats in the Port of Rangoon.—*Brit. Med. Jl.*, 1913. Aug. 23. pp. 439-441.
- GOFF** (A. P.). Bubonic Plague in Manila.—*Jl. Amer. Med. Assoc.*, 1913. June 28. Vol. 60. No. 26, pp. 2042-2043.
- GRUBBS** (S. B.) & **HOLSENDOFF** (B. E.). Fumigation of Vessels for the Destruction of Rats.—*U.S. Public Health Rep.*, 1913. June 20. Vol. 28. No. 25, pp. 1266-1274.
- HEISER** (Victor G.). The Outbreak of Plague in Manila during 1912. The Insidious Beginning, with a Discussion of Probable Factors concerned in its Introduction.—*Philippine Jl. of Science*, Section B. Trop. Med., 1913. April. Vol. 8. No. 2, pp. 109-115. With 1 map.
- . The Rats of our Cities. What becomes of the Carcasses of Rats dying Natural Deaths?—*U.S. Public Health Repts.*, 1913. July 25. Vol. 28. No. 30, pp. 1553-1554.
- JOURDAN** (Victor J. P.). Bubonic Plague: its History and Prevention.—*Monthly Cyclopaedia & Med. Bull.*, 1913. May. New Ser. Vol. 16. No. 5. (Old Ser. Vol. 27), pp. 270-274.
- KITASATO** (S.). On the Value of the Search for Rat-Fleas in the Detection of Plague.—*Trans. xvii Intern. Congress of Med., London*, 1913. Section xxi, Trop. Med. & Hyg. Part 1, pp. 1-7.
- LEMAN** (Issac Ivan). The Treatment of the Plague.—*Southern Med. Jl.*, 1913. July 1. Vol. 6. No. 7, pp. 446-448.
- LISTON** (W. Glen). Plague.—*Trans. xvii Intern. Congress of Med., London*, 1913. Section xxi, Trop. Med. & Hyg. Part 1, pp. 9-23.
- LURZ** (Richard). Eine Pestepidemie am Kilimandscharo im Jahre 1912.—*Arch. f. Schiffs- u. Trop. Hyg.*, 1913. Sept. Vol. 17. No. 17, pp. 593-599.
- MEYER** (Carlos). Instituto Bacteriologico do Estado de S. Paulo. Relatório sobre a administração e os trabalhos do Instituto durante o anno de 1912.—*Revista Med. de S. Paulo*, 1913. Feb. 15. Vol. 16. No. 3, pp. 56-61. [Bubonic plague, p. 58.]
- MONTÉL**. Lésions Bucco-Pharyngées dans la Peste. [Clinique d'Outre-Mer].—*Ann. d'Hyg. et Med. Colon*, 1913. July-Aug.-Sept. Vol. 16. No. 3, pp. 779-781.
- PIRAS** (L.). Die Präzipitinreaktion als diagnostisches Mittel der Pest.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Sept. 27. Vol. 71. No. 1, pp. 69-80. With 1 text-fig.
- ROBINSON** (George H.). The Rats of Providence and their Parasites.—*Amer. Jl. of Public Health*, 1913. Aug. Vol. 3. No. 8, pp. 773-776.
- DE SOUSA JUNIOR** (Antonio). Algumas considerações sobre a Peste Bubonica.—*Medicina Contemporanea*, 1913. June 15. Vol. 31. No. 24, pp. 185-187.
- STRICKLAND** (C.). The Bionomics of the Rat-Flea. [Correspondence].—*Brit. Med. Jl.*, 1913. Aug. 16. p. 435.
- SUDHOFF** (Karl). Syphilis und Pest in München am Ende des 15. und zu Anfang des 16. Jahrhunderts. (Eine Urkundenstudie).—*München. Med. Wochenschr.*, 1913. July 1. Vol. 60. No. 26, pp. 1439-1443.
- WU LIEN TEH** [G. L. Tuck]. Investigations into the Relationship of the Tarbagan (Mongolian Marmot) to Plague.—*Lancet*, 1913. Aug. 23. pp. 529-535. With 4 text-figs.: and *Jl. Trop. Med. & Hyg.*, 1913. Sept. 15. Vol. 16. No. 18, pp. 275-280.



## Relapsing Fever (and Spirochaetosis).

- BALFOUR** (Andrew). A Contribution to the Life-History of *Spirochaetes*. A Reply to Dr. Gleitsmann.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 4. Vol. 70. Nos. 3/4, pp. 182-185.
- CONSEIL** (E.). La Fièvre Récurrente Nord-Africaine. (Etude Clinique sur Cent Soixante Cas.)—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 38-66.
- . Chimiothérapie de la Fièvre Récurrente.—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 67-87.
- GLEITSMANN**. Beitrag zur Entwicklungsgeschichte der Spirochäten (Borrelien). Ein Schlusswort zu Balfours Erwiderung.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 4. Vol. 70. Nos. 3/4, pp. 186-187.
- KUSUNOKI** (F.). Experimentelle Untersuchungen über Heredo-Immunität bei afrikanischer Recurrens und über den etwaigen Einfluss von Immunitätsvorgängen auf die Wirksamkeit eines chemotherapeutischen Mittels.—*Zeitschr. f. Chemotherapie*, I. Teil., Orig., 1913. Vol. 2. No. 1, pp. 11-22.
- LAMOUREUX** (A.). La Fièvre Récurrente de Madagascar. Considérations cliniques. Le Spirochète dans l'Organisme humain. Essai de Traitement par le 606.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 523-533. With 7 curves.
- MACKIE** (F. P.). The Body Louse (*Pediculus vestimenti*) as a Disease Carrier. The Body Louse as a Carrier of Relapsing Fever.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras*, Nov. 18, 19, 20, 1912. pp. 281-289. (1913. Simla: Govt. Central Branch Press.)
- MARCHEUX** (E.) & **COUVY** (L.). Argas et Spirochètes. (Premier Mémoire.) Les Granules de Leishman.—*Ann. Inst. Pasteur*, 1913. June. Vol. 27. No. 6, pp. 450-480. With 15 text-figs.
- & ———. Argas et Spirochètes. (Deuxième Partie.) Le Virus chez l'Acarien.—*Ann. Inst. Pasteur*, 1913. Aug. 25. Vol. 27. No. 8, pp. 620-643.
- MOUZELS** (P.). La Fièvre Récurrente au Tonkin et plus particulièrement à Hanoï pendant les Epidémies de 1911 et 1912.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 249-282.
- NICOLLE** (Ch.) & **BLAIZOT** (L.). Courte durée de l'Immunité dans la Fièvre Récurrente Expérimentale. Valeur nulle de l'Epreuve de la Vaccination croisée pour la Distinction des Spirochètes du Groupe *Obermeieri*.—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 31-36.
- , ———, & **CONSEIL** (E.). L'Epidémie Tunisienne de 1912 et la Demonstration Expérimentale de la Transmission de la Fièvre Récurrente par les Poux. — *Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 1-30.
- SANGIORGI** (Giuseppe). Spirochetosi della Cavia. — *Pathologica*, 1913. July 15. Vol. 5. No. 113, pp. 428-430. With 3 text-figs.
- SERGEANT** (Edm.). Infection de Fièvre Récurrente par les Muqueuses chez l'Homme.—*Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, p. 185.
- SOFER** (Willard B.). A Case of *Spirillum* Infection.—*Arch. Internal Med.*, 1913. Sept. 15. Vol. 12. No. 3, pp. 273-275.
- STEEN** (R.) & **TOWNSEND** (R. S.). Relapsing Fever in Bulandshahr District, U.P., 1912-1913.—*Indian Med. Gaz.*, 1913. Sept. Vol. 48. No. 9, pp. 338-341. With 6 temperature charts.
- WINOCOUROFF** (J.). Rückfallfieber bei Kindern in Odessa. (Eigene Beobachtungen vom Jahre 1890-1910.)—*Arch. f. Kinderheilkunde*, 1913. May 24. Vols. 60/61. (Festschrift f. Adolf Baginsky.) pp. 777-789. With 3 curves.

## Skin, Tropical Diseases of the.

- ARLO (J.).** Pied de Madura avec Envahissement du Triangle de Scarpa et de la Partie inférieure de la Paroi abdominale. — *Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 485-487; and *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 440-442.
- BRAULT (J.).** Note sur les Cultures de *Madurella mycetomi*. — *Bull. Soc. Path. Exot.*, 1913. June. Vol. 6. No. 6, pp. 407-409.
- BREINL (Anton).** Prevalence of Keratosis followed by Epithelioma of the Skin in North-western Queensland. — *Australian Inst. of Trop. Med. Report for the Year 1911*. pp. 24-26.
- BUARD & CREIGHTON.** Préparations de Pus d'Ulcère Phagédénique des Pays Chauds. (Société Anatomo-Clinique de Bordeaux. Séance du 19 Mai 1913.) — *Jl. de Méd. de Bordeaux*, 1913. Sept. 14. Vol. 84. No. 37, p. 597.
- CARINI (A.).** Sopra un Caso di Micetoma della Guancia. — *Giorn. Italiano d. Malattie Veneree e d. Pelle*, 1913. No. 2, 3 pp. With 2 figs.
- CHIPMAN (Ernest Dwight).** The Newer Cutaneous Mycoses. — *Jl. Amer. Med. Assoc.*, 1913. Aug. 9. Vol. 61. No. 6, pp. 407-412. With 3 text-figs.
- LOP (M.).** Un Cas Type de Pied de Madura. Amputation. Guérison. — *Gaz. des Hôpît. Civils et Militaires*, 1913. July 8. Vol. 86. No. 77, p. 1255.
- TANGANELLI (F.).** Un Caso di Noma in un Soldato reduce dalla Libia. — *Ann. di Med. Nav. e Colon.*, 1913. Vol. 1. Nos. 5/6, pp. 543-545.

## Sleeping Sickness (and other Trypanosomiasis).

- ALEXIEFF (A.).** A propos du Corpuscule Préblépharoplastique chez les Trypanosomes. (Réponse à M. Roudsky.) — *Arch. f. Protistenkunde*, 1913. Sept. 12. Vol. 30. No. 3, pp. 322-325.
- AOKI (K.) & KODAMA (H.).** Beitrag zur Frage der Immunisierung mit abgetöteten Trypanosomen. — *Zeitschr. f. Immunitätsforsch. u. experim. Therapie*, 1. Teil., Orig., 1913. July 26. Vol. 18. No. 6, pp. 693-700.
- ARAGAO (Henrique de Beaurepaire).** Nota sobre as Schizogonias e Gametogonias dos Trypanosomos. *Brazil Medico*, 1913. July 15. Vol. 27. No. 27, pp. 271-272.
- AUSTEN (E. E.).** The Present Position of the Problem of Big Game, Tsetse Flies, and Sleeping Sickness. — *Jl. Soc. for Preservation of Wild Fauna of the Empire*, 1913. Vol. 6, pp. 57-71.
- BERNARD.** Relation d'une Tournée Médicale faite dans la Région du Djéma et du M'Bomou (Oubangui-Chari) Avril et Mai, 1912. — *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 321-331.
- BONGER (C.).** Ueber die Morphologie und das Verhalten der von P. Behn in deutschen Rindern nachgewiesenen Trypanosomen bei künstlicher Infektion. — *Zeitschr. f. Hygiene u. Infektionskr.*, 1913. July 17. Vol. 75. No. 1, pp. 101-117. With 1 plate.
- BOURRET.** Recherches sur le Parasitisme Intestinal, la Dysenterie et la Maladie du Sommeil à Saint-Louis (Sénégal). — *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 283-307.
- CARPANO (M.).** Trypanosomen vom Typus des *Tr. Theileri* in den Rindern der Kolonie Erythraea. — *Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 28. Vol. 70. Nos. 5/6, pp. 209-217. With 1 plate.
- CORYNDON (R. T.).** Tsetse Fly and Big Game. — *Jl. Soc. for Preservation of Wild Fauna of the Empire*, 1913. Vol. 6, pp. 41-56.

- DA COSTA (Bernardo F. Bruto). *Trabalhos sobre a Doença do Sono. Saneamento, Estatística Serviços Hospitalares e Brigada Oficial na Ilha do Príncipe.* 78 pp. With 3 plates. (1913. Lisbon: Typographia a Editora Limitada.)
- DIAS (Ezequiel). *Molestia de Carlos Chagas. Estudos hematológicos.—Revista Med. de S. Paulo, 1912. Dec. 31. Vol. 15. No. 24, pp. 484-494.*
- DUNBAR-BRUNTON (J.). *Sleeping Sickness and Big Game. [Correspondence].—Brit. Med. J., 1913. July 19. pp. 150-151.*
- ECKARD (B.). *Ueber Schlafkrankheit.—Arch. f. Schiffs- u. Trop.-Hyg., 1913. July. Vol. 17. No. 14, pp. 494-497.*
- FISCHER (W.). *Über das Vorkommen von Kernverlagerungen bei Trypanosoma brucei.—Arch. f. Schiffs- u. Trop.-Hyg., 1913. Sept. Vol. 17. No. 18, pp. 621-626. With 16 text-figs. and 1 curve.*
- FISKE (William F.). *The Bionomics of Glossina: A Review with Hypothetical Conclusions.—Bull. Entomol. Research, 1913. Sept. Vol. 4. Pt. 2, pp. 95-111.*
- FLEMING (A. M.). *Trypanosomiasis in Southern Rhodesia.—Trans. Soc. Trop. Med. & Hyg., 1913. July. Vol. 6. No. 8, pp. 298-310. With a map.*
- FRY (W. B.) & RANKEN (H. S.). (With a Note on Methods by H. G. PLIMMER.) *Further Researches on the Extrusion of Granules by Trypanosomes and on their Further Development.—Jl. R. Army Med. Corps, 1913. Aug. Vol. 21. No. 2, pp. 137-155. With 3 plates.*
- HECKENROTH (F.) & BLANCHARD (M.). *Réaction de Fixation, en Présence d'Antigène Syphilitique, dans la Syphilis, le Pian, la Trypanosomiasse et l'Ulçère phagédénique au Congo français.—Compt. Rend. Acad. Sciences, 1913. Sept. 1. Vol. 157. No. 9, pp. 437-440; and Caducée, 1913. Sept. 20. Vol. 13. No. 18, p. 244.*
- HOLMES (J. D. E.). *Some Cases of Surra treated in the Field and in the Laboratory during the Autumn of 1911.—Memoirs of the Dept. of Agriculture in India. Veterinary Series. 1913. Aug. Vol. 2. No. 1, pp. 1-31.*
- JOHNS (Foster M.). *On the Adult Forms of Trypanosoma americanum in Naturally Infected Animals.—Amer. Jl. Trop. Diseases & Preventive Med., 1913. July. Vol. 1. No. 1, pp. 49-59. With 1 coloured plate.*
- KERANDEL (J.). *Trypanosomes et Leucocytozoon observés chez des Oiseaux du Congo.—Ann. Institut Pasteur, 1913. June. Vol. 27. No. 6, pp. 421-439. With 2 plates.*
- KLEINE (F. K.) & ECKARD (B.). *Ueber die Bedeutung der Haustiere und des Wildes für die Verbreitung der Schlafkrankheit.—Zeitschr. f. Hygiene u. Infektionskr., 1913. July 17. Vol. 75. No. 1, pp. 118-122.*
- & FISCHER (W.). *Schlafkrankheit und Tsetsefliegen (II. Mitteilung).—Zeitschr. f. Hyg. u. Infektionskr., 1913. Aug. 27. Vol. 75. No. 2, pp. 375-382.*
- KOHL-YAKIMOFF (Nina), YAKIMOFF (W. L.), & BEKENSKY (P. W.). *Le Trypanosome des Bovidés (Tr. theileri ou du Type voisin) en Russie d'Europe.—Bull. Soc. Path. Exot., 1913. June. Vol. 6. No. 6, pp. 433-434.*
- , —, & SCHOKHOB (N. J.). *Le Trypanosome des Bovidés (Tr. theileri ou du Type voisin) au Turkestan.—Bull. Soc. Path. Exot., 1913. June. Vol. 6. No. 6, p. 434.*
- KOLLE (W.), HARTOCH (O.), ROTHERMUNDT (M.), & SCHÜRMANN (W.). *Chemotherapeutische Experimentalstudien bei Trypanosomeninfektion.—Zeitschr. f. Immunitätsforsch. u. experim. Therapie., 1. Teil., Orig., 1913. Aug. 5. Vol. 19. No. 1, pp. 66-97.*

- KOPKE** (Ayres). Traitement de Quelques Cas de Trypanosomiase Humaine par le Salvarsan et le Neosalvarsan.—*Med. Contemporanea*, 1913. Sept. 14. Vol. 31. No. 37, pp. 289-292.
- LAVERAN** (A.) & **ROUDSKY** (D.). Le Galyl dans les Trypanosomiasés.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 502-505.
- LURZ** (R.). Ein Mittel von L. Brieger und M. Krause zur Behandlung der Trypanosomen im menschlichen Organismus.—*Arch. f. Schiff- u. Trop.-Hyg.*, 1913. Sept. Vol. 17. No. 18, pp. 636-640.
- MACFIE** (J. W. Scott). On the Morphology of the Trypanosome (*T. nigeriense*, n. sp.) from a Case of Sleeping Sickness from Eket, Southern Nigeria.—*Ann. Trop. Med. & Parasit.*, 1913. Aug. 11. Vol. 7. No. 3A, pp. 339-356.
- MACKIE** (F. P.). The Investigation of Protozoal Diseases with Special Reference to the Differentiation of Trypanosomes.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 271-280. (1913. Simla: Govt. Central Branch Press.)
- MOISER** (Bernard). Notes on a Few Photographs illustrating the Haunts and Habits of *Glossina tachinoides* in Bornu, Northern Nigeria.—*Bull. Entomol. Research*, 1913. Sept. Vol. 4. Pt. 2, p. 145. With 5 plates.
- MORGENROTH** (J.) & **TUGENDREICH** (J.). Aethylhydrocuprein und Salicylsäure als Adjuvantien des Salvarsan.—*Berlin Klin. Wochenschr.*, 1913. No. 26. 16 pp.
- NEAVE** (Sheffield). Sleeping Sickness and Big Game. [Correspondence].—*Brit. Med. J.*, 1913. July 19. p. 150.
- NEIVA** (Arthur). Da Transmissao do *Trypanosoma cruzi* pela *Triatoma sordida* Stal.—*Brazil Medico*, 1913. Aug. 8. Vol. 27. No. 30, p. 309.
- NYASALAND PROTECTORATE**. Sleeping Sickness Diary. Part 20. 1913.
- OEHLER** (R.). Zur Gewinnung reiner Trypanosomenstämme.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 29. Vol. 70. Nos. 1/2, pp. 110-111.
- OFFERMANN**. Zur Frage der Immunität bei Trypanosomenkrankheiten.—*Zeitschr. f. Veterinärkunde*, 1913. Vol. 25. No. 7, pp. 299-301.
- PRINGAULT** (E.). Contribution à l'Etude des Trypanosomes de l'Afrique Mineure. *Arch. Inst. Pasteur Tunis*, 1913. Nos. 1 2, pp. 119-120.
- RANKEN** (H. S.). A Preliminary Report on the Treatment of Human Trypanosomiasis and Yaws with Metallic Antimony (Plimmer).—*Jl. R. Army Med. Corps*, 1913. Sept. Vol. 21. No. 3, pp. 261-281.
- RAVENNA** (Ettore). Lesioni Endocardiche nella Tripanosomiasi sperimentale.—*Archivio per le Scienze Mediche*, 1913. July 10. Vol. 37. No. 3, pp. 236-249.
- RÉCAMIER**. Un Cas de Trypanosomiase observé chez un Tirailleur indigène à Fort-Lamy.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 442-443.
- RITS** (H.). Chemotherapeutische Versuche mit "Trypasafrol." (Zum Teil nach Versuchen von Frl. F. Leupold.)—*Berlin Klin. Wochenschr.*, 1913. July 28. Vol. 50. No. 30, pp. 1387-1389.
- ROMANESCU** (R.). Sulle Iniezioni Endovenose di Bleu di Metilene nella Tripanosomiasi Sperimentale.—*Archivio per le Scienze Mediche*, 1913. July 10. Vol. 37. No. 3, pp. 250-257.
- RONDONI** (Pietro) & **GORETTI** (Guido). Studien über Schutzimpfung gegen experimentelle Naganainfektion.—*Zeitschr. f. Immunitätsforsch. u. Experim. Therapie*, 1. Teil, Orig., 1913. July 17. Vol. 18. No. 5, pp. 491-515.

- RONDONI (Pietro) & GORETTI (Guido).** Ricerche Sperimentali sul Nagana. —*Lo Sperimentale*, 1913. Aug. 16. Vol. 67. No. 4, pp. 427-453.
- ROSENTHAL (Felix).** Untersuchungen über die Genese des Rezidivs bei der experimentellen Trypanosomeninfektion.—*Zeitschr. f. Hyg. u. Infektionskr.*, 1913. June 26. Vol. 74. No. 3, pp. 489-538.
- ROUDSKY (D.).** Réponse à Monsieur Alexeieff.—*Arch. f. Protistenkunde*, 1913. Sept. 12. Vol. 30. No. 3, pp. 326-327.
- SCHERN (Kurt) & CITRON (Heinrich).** Ueber Lävulosurie, sowie neuartige Serum- und Leberstoffe bei Trypanosomiasis. — *Deut. Med. Wochenschr.*, 1913. July 10. Vol. 39. No. 28, pp. 1356-1357.
- SCHILLING (Claus) & RONDONI (Pietro).** Ueber Trypanosomen-Toxine und Immunität.—*Zeitschr. f. Immunitätsforsch. u. experim. Therapie.*, 1. Teil., Orig., 1913. July 26. Vol. 18. No. 6, pp. 651-665.
- SHIRCORE (J. O.).** Nyasaland Trypanosome Fever.—*Ann. Med. Report on Health & Sanitary Condition of the Nyasaland Protectorate for year ended 31st March*, 1913. pp. 72-77. (London: Printed by Waterlow & Sons, Ltd.)
- STRACHAN (Henry).** West African Notes. (2) Notes on the Bites of the Tsetse-Fly (*Glossina palpalis*). — *Jl. Trop. Med. & Hyg.*, 1913. July 15. Vol. 16. No. 14, p. 214.
- TAUTE (M.).** Untersuchungen über die Bedeutung des Grosswildes und der Haustiere für die Verbreitung der Schlafkrankheit im Nyassaland (*Trypanosoma rhodesiense*).—*Arbeit. u.d. Kais. Gesundheitsamt.*, 1913. Vol. 45. No. 1, pp. 102-112.
- TODD (J. L.).** Big Game and Sleeping Sickness. [Correspondence].—*Brit. Med. Jl.*, 1913. July 26. p. 207.

## Typhus Fever.

- MARKL.** Beitrag zur serologischen Diagnose des Flecktyphus.—*Wien. Klin. Wochenschr.*, 1913. July 24. Vol. 26. No. 30, pp. 1234-1235.

## Undulant Fever.

- GARDNER-MEDWIN (F. M.).** A Case of Malta Fever.—*Liverpool Medico-Chirurg. Jl.*, 1913. July. No. 64, pp. 386-394.
- HITCHENS (A. Parker).** Serums and Vaccines in the Prevention and Treatment of Undulant Fever.—*Amer. Jl. Trop. Dis. & Preventive Med.*, 1913. Sept. Vol. 1. No. 3, pp. 228-245.
- MARTEL, TANON & CHÉTIEN.** La Valeur de l'Agglutination du *Micrococcus melitensis* par le Sérum sanguin en particulier chez les Chèvres.—*Presse Méd.*, 1913. Aug. 20. No. 68, pp. 685-686.
- MEREU (Francesco).** Febbre di Malta nella Miniera Argentiera (Sassari). —*Policlínico, Sez. prat.*, 1913. June 29. Vol. 20. No. 26, pp. 947-949.
- MOHLER (J. R.) & EICHORN (A.).** Malta Fever, with special Reference to its Diagnosis and Control in Goats.—*U.S. Dept. of Agriculture. 28th Annual Report of the Bureau of Animal Industry for the year 1911.* pp. 119-136. With 4 plates. 1913. Washington: Govt. Printing Office.
- TROTTA (Guido) & CANTIERI (Collatino).**—Ueber zwei Fälle von Eiterung bei Maltafieber, welche Senkungsabszesse im Gefolge von Malum Potti vortäuschten. — *Wien. Klin. Wochenschr.*, 1913. Aug. 23. Vol. 26. No. 35, pp. 1895-1898.
- & —. I Processi Suppurativi nella Febbre Mediterranea. — *Riv. Crit. Clin. Med.*, 1913. Sept. 13. Vol. 14. No. 37, pp. 577-584; and Sept. 20. No. 38, pp. 593-598.

- VIGANÒ (Luigi). Die Thermoprazipitinreaktion des Maltafiebers.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Aug. 4. Vol. 70. Nos. 3/4, pp. 200-202.
- . Le Termoprecipitine del Micrococco melitense.—*Giorn. d. R. Soc. Ital. d'Igiene*, 1913. Aug. 31. Vol. 35. No. 8, pp. 337-340, and *Polichinico*, Sez. Med., 1913. Sept. Vol. 20. No. 9, pp. 430-432

## Yaws.

- BAERMANN (G.) & HEINEMANN (H.). Die Intra kutaneaktion bei Syphilis und Frambosie.—*Munchen Med Wochenschr.*, 1913. July 15 Vol. 15. Vol. 60. No. 28, pp. 1537-1542
- KLOPPERS (J. W. E. R. S.). Opmerkingen over Framboesia.—*Geneesk Tijdschr. v Nederl-Indië*, 1913. Vol. 53. No. 1. pp 18-31
- RADLOFF Frambosie und Salvarsan.—*Arch f Schiffs- u. Trop Hyg*, 1913. July. Vol 17. No. 13, pp. 459-460.
- RANKEN (H. S.). A Preliminary Report on the Treatment of Human Trypanosomiasis and Yaws with Metallic Antimony (Plimmer) *Jl. R. Army Med. Corps*, 1913. Sept Vol 21. No 3 pp 261-281.
- SCHERSCHMIDT (Arthur). Erfahrungen mit Joda bei Frambosie *Arch f Schiffs- u Trop Hyg*, 1913 Vol 17 No. 16, pp 552-559

## Yellow Fever.

- HOWLETT (L M.). *Stegomyia fuscata* the Yellow Fever Mosquito *Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912* p. 205 (1913 Simla Govt. Cential Branch Press)

See also MISCELLANEOUS: Biting Arthropods and Ticks.

## Miscellaneous.

(CLIMATIC BUBO, RAT BITE DISEASE. SPRUE, VERRUGA PERUVIANA).

- ASHFORD (Bailey K.). Notes on Sprue in Porto Rico and the Results of Treatment by Yellowed Santonin. — *Amer. Jl. Trop. Diseases & Preventive Med.*, 1913. Aug. Vol. 1. No. 2, pp. 146-158.
- BREINL (Anton). Two Cases of " Climatic Bubo. " *Australian Inst. of Trop. Med. Report for the Year 1911*, pp. 27-29
- NICHOLSON (Frank). A further Case of Rat-Bite Disease — *Practitioner*, 1913. Sept. Vol. 91. No. 3. [No. 543], pp. 429-430.
- DA ROCHA-LIMA (H.). Zur Histologie der Verruga peruviana.— *Verhandlungen der Deut. Pathologischen Gesellschaft*, 1913. pp. 409-416. With 1 plate and 2 text-figs.
- TOWNSEND (Ch. H. T.). La Titira es Transmisora de la Verruga.—*Cronica Medica*, 1913. June 30. Vol. 30. No. 588, pp. 210-211.
- . Progress in the Study of Verruga Transmission by Bloodsuckers — *Bull. Entomol. Research*, 1913. Sept. Vol. 4. Pt. 2, pp. 125-128. With 3 plates.

## UNCLASSSED.

- AUMANN. Reiseskizzen aus Mittelbrasilien.—*Munchen Med. Wochenschr.*, 1913. Aug. 26. Vol. 60. No. 34, pp. 1888-1889.
- BOBEAU (G.). Importance des Affections Mycosiques en Cochinchine.— *Compt. Rend. Soc. Biol.*, 1913. July 18. Vol. 75. No. 26, pp. 69-70.

- CORDIER & SÉGUIN.** Observation de Mycose Pulmonaire simulant la Tuberculose. — *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 449-451.
- DEBKS (W. E.) & BAETS (W. G.).** An Analysis of Five Hundred Fatal Medical Cases in the Tropics. With the Clinical Diagnosis in the Light of Autopsy Findings.—*New York Med. J.*, 1913. Sept. 6. Vol. 98. No. 10. [Whole No. 1814], pp. 462-465.
- FOLLEY (H.).** Les Ophtalmies contagieuses dans le Sahara Oranais.—*Ann. d'Oculistique*, 1913. April. 12 pp.
- FROES (J. A. G.).** Hemodiagnostico nos Tropiccos.— *Brazil Medico*, 1913.
- GASTALDI (G.).** Ricerche sul Potere Lipolitico del Siero in Varie Forme di Malattia.—*Riv. Crit. Clin. Med.*, 1913. Sept. 20. Vol. 14. No. 38, pp. 599-601.
- GIEMSA (G.).** Paraffinöl als Einschlussmittel für Romanowsky-Präparate und als Konservierungsflüssigkeit für ungefärbte Trockenausstriche. — *Centr. bl. f. Bakt.*, 1. Abt., Orig., 1913. Sept. 13. Vol. 70, pp. 444-446.
- HAPKE.** Maximale Eosinophilie bei Tropenschwellung.— *Arch. f. Schiffs- u. Trop. Hyg.*, 1913. July. Vol. 17. No. 13, p. 462.
- HEWLETT (R. Tanner).** Review of Tropical Diseases. *Practitioner*, 1913. Aug. Vol. 91. No. 2. (No. 542), pp. 218-226.
- HOLST (Axel) & FRÖLICH (Theodor).** Ueber experimentellen Skorbut. II. Mitteilung. Weitere Untersuchungen über das Konservieren und Extrahieren der spezifischen Bestandteile der antiskorbutischen Nahrungsmittel. — *Zeitschr. f. Hyg. u. Infektionskr.*, 1913. Aug. 27. Vol. 75. No. 2, pp. 334-344.
- HUNTEMÜLLER.** Filtrierbare Virusarten. — *Zeitschr. f. Chemotherapie*, I. Teil., Orig., 1913. Vol. 2. No. 1, pp. 56-70. With 9 plates.
- KOUN.** Fonctionnement du Deuxième Service de l'Hopital de Hué pendant l'Année 1911.— *Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 416-436.
- LESK (Robert).** Einiges über Erkrankungen der Gallenwege und Leber. Ein tropenchirurgischer Beitrag.— *Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 3, pp. 386-415.
- MARSHALL & MEERWEIN (Werner).** Über das leukozytäre Blutbild, einschliesslich Verschiebung der Neutrophilen, bei wilden Eingeborenen von Neuguinea. *Folia Haematologica*, 1913. Vol. 15. pp. 229-236.
- MEDIZINAL-BERICHT** über die Deutschen Schutzgebiete Deutsch-Ostafrika, Kamerun, Togo, Deutsch Südwestafrika, Deutsch-Neuguinea, Karolinen, Marshall- und Palau-Inseln und Samoa für das Jahr 1910/11.—xii.+808 pp. With 5 plates and 49 figs. (1913. Berlin: Ernst Siegfried Mittler & Sohn.)
- MOUCHET.** Notes Anatomiques et Médicales sur la Pathologie du Moyen Congo. — *Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Oct. Vol. 17. No. 19, pp. 657-669.
- NÄGELE.** Eine nomaähnliche Erkrankung bei einem Bastardkind in Südwestafrika.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. July. Vol. 17. No. 13, pp. 464-466. With 1 text-fig.
- NOC (F.).** Le Preventorium Colonial de Fort-de-France (Martinique). Description et Fonctionnement.—*Mémoire couronné par la Société de Médecine publique et de Génie sanitaire*, 1913. 32 pp. With 10 text-figs. (Paris: Masson et Cie.)
- OTIS (Elmer F.).** Diseases of Porto Rico.—*Jl. Amer. Med. Assoc.*, 1913. Sept. 27. Vol. 61. No. 13, Part 1, pp. 1031-1034.
- PFISTER (E.).** Die Steinkrankheit bei der Negerrasse.—*Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Sept. Vol. 17. No. 17, pp. 599-604.
- REMLINGER (P.).** Essai de Nosologie Marocaine.—*Ann. d'Hyg. Publique et de Méd. Légale*, 1913. Aug. 4<sup>e</sup> ser. Vol. 20. pp. 129-167.

- RENAULT.** Fonctionnement du Premier Service de l'Hôpital de Hué pendant l'Année 1911.—*Ann. d'Hyg. et Méd. Colon.*, 1913. April-May-June. Vol. 16. No. 2, pp. 400-416.
- ROBERTSON (J. C.).** A Short Note on the Relation between the Seasonal Birth- and Death-Rate Curves.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 95-96. With a curve. (1913. Simla: Govt. Central Branch Press.)
- ROGERS (L.).** Gleanings from the Calcutta Post-Mortem Records. No. vii. Diseases of the Nervous System.—*Indian Med. Gaz.*, 1913. Aug. Vol. 48. No. 8, pp. 293-297.
- RUFFO (Albino).** Di un Nuovo Metodo di Colorazione delle Cellule granulose (Mastzellen). — *Gazz. Internaz. Med. Chirur. Igiene*, 1913. June 28. No. 26, pp. 609-610.
- SCHERSCHMIDT (Arthur).** Eine Vereinfachung der intravenösen Salvarsan-injektion. — *Arch. f. Schiffs- u. Trop.-Hyg.*, 1913. Vol. 17. No. 16, pp. 552-559.
- SPLENDORÉ (A.).** Un Affezione Micotica con Localizzazione nella Mucosa della Bocca, osservata in Brasile, determinate da Funghi appartenenti alla Tribù degli Exoascei (*Zygonema brasiliense*, n. sp.).—*Estratto del volume "In onore del Prof. Angelo Celli nel 25° anno di insegnamento."* pp. 421-458. With 6 plates. 1912 Roma. Tipografia Nazionale di G. Bertero E. C.
- STEVENSON (E. Sinclair).** Splenomegaly.—*Brit. Med. J.*, 1913. Oct. 4. pp. 847-849. With 3 text-figs.
- ZUR VERTH.** Die tropische Schwärsucht.—*Deut. Militärarzt Zeitschr.*, 1913. Aug. 5. Vol. 42. No. 15, pp. 584-590.
- WESTCOTT (Sinclair).** Flies and Disease in the British Army. *Jl. State Med.*, 1913. Aug. Vol. 21. No. 8, pp. 480-498.
- ZIMMANN (H.).** Zur Pathogenese, Diagnose und Prophylaxe der Tuberkulose in den Tropen.—*Centralbl. f. Bakt.* 1. Abt., Orig., 1913. Aug. 4. Vol. 70. Nos. 3/4, pp. 118-141.
- Biting Arthropods and Ticks.**
- BISHOPP (F. C.) & WOOD (H. P.).** The Biology of some North American Ticks of the Genus *Dermacentor*.—*Parasitology*, 1913. July. Vol. 6. No. 2, pp. 153-187. With 3 plates and 1 map.
- COOLEY (R. A.).** Notes on Little Known Habits of the Rocky Mountain Spotted Fever Tick (*Dermacentor venustus* Banks). With Discussion.—*Jl. Economic Entomology*, 1913. Feb. Vol. 6. No. 1, pp. 93-96.
- FRICKS (L. D.).** Rocky Mountain Spotted (or Tick) Fever. Sheep Grazing as a possible means of controlling the Wood Tick (*Dermacentor Andersoni*) in the Bitter Root Valley.—*U.S. Public Health Repts.*, 1913. Aug. 8. Vol. 28. No. 32, pp. 1647-1653.
- HORNE (J. H.).** Notes on Distribution and Habits of *Stegomyia* Mosquitoes in Madras. — *Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 197-199. (1913. Simla: Govt. Central Branch Press.)
- HOWLETT (L. M.).** Insect Psychology.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* p. 33. (1913. Simla: Govt. Central Branch Press.)
- LALOR (N. P. O'Gorman).** A Brief Report of the *Stegomyia* Survey in the Principal Ports of Burma.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 201-203. (1913. Simla: Govt. Central Branch Press.)
- LEGENDRÉ (J.).** Note sur les *Stegomyias* du Tonkin.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 511-513.



- LEGENDRE (J.). Destruction des Culicines à l'Aide du Gîte-Piège.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 513-514.
- LISTON (W. Glen). A Stegomyia Survey of the City and Island of Bombay.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 187-188. (1913. Simla: Govt. Central Branch Press.)
- MACGILCHRIST (A. C.). Progress Report—Stegomyia Survey—Port of Calcutta.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 193-196. (1913. Simla: Govt. Central Branch Press.)
- MEASKAR (K. S.). Stegomyia in Karachi.—*Proceedings of the Third Meeting of the General Malaria Committee held at Madras, Nov. 18, 19, 20, 1912.* pp. 189-192. (1913. Simla: Govt. Central Branch Press.)
- PITTALUGA (G.). El "Je-jén": Un nuevo Diptero hematófago de la Costa Occidental de Africa (Guinea Espanola). *Oecacta hostilisima*, n. sp.—*Bol. R. Soc. Espanola de Historia natural*, 1912. Dec. pp. 591-600. With 1 plate.
- STRACHAN (Henry). West African Notes. (1) Note on a Portable Insect-Proof Room.—*Jl. Trop. Med. & Hyg.*, 1913. July 15. Vol. 16. No. 14, p. 214.
- WEISS (A.). Seconde addition au Catalogue des Arthropodes Piqueurs de l'Ile de Djerba.—*Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, p. 116.

# Protozoology (excluding Trypanosomes and Amoebae).

- BREINL (Anton). Parasitic Protozoa encountered in the Blood of Australian Native Animals.—*Australian Inst. of Trop. Med. Report for the Year 1911.* pp. 30-38.
- COMINOTTI (L.). Über Sarkosporidin.—*Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. June 4. Vol. 69. No. 4, pp. 264-271.
- FANTHAM (H. B.). *Sarcocystis colii*, n. sp. A Sarcosporidian occurring in the Red-Faced African Mouse Bird, *Colius erythromelon*.—*Proc. Cambridge Philosophical Soc.*, 1913. July 21. Vol. 17. Pt. 3, pp. 221-224. With 1 plate.
- FLU (P. C.). Over de z.g.n. Kurloffschamen in de mononukleaire Bloedcellen van *Cavia cobaya*.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 52. No. 6, pp. 679-702. With 1 plate.
- . Bemerkungen zu der Obenstehenden "Berichtigung" Schilling-Torgau's.—*Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 2, pp. 359-364.
- FRANCHINI (G.). Nuovo Contributo allo Studio dell' *Haemcystozoon brasiliense*.—*Ann. di Med. Nav. e Colon.*, 1913. Vol. 1. Nos. 5/6, pp. 477-480.
- LAVERAN (A.) & FRANCHINI (G.). Infections expérimentales de la Souris par *Herpetomonas ctenocephali*.—*Compt. Rend. Acad. Sciences*, 1913. Sept. 1. Vol. 157. No. 9, pp. 423-426.
- LEGER (Marcel). Hématozoaires d'Oiseaux de la Corse.—*Bull. Soc. Path. Exot.*, 1913. July. Vol. 6. No. 7, pp. 515-523.
- MACEDO (Astrogildo). Sobre o Ciclo evolutivo de *Schizocystis spinigeri*, n. sp. Gregarina do Intestino de uma Especie de Spiniger.—Ueber den Entwicklungskreis einer Gregarine, *Schizocystis spinigeri* aus Spiniger spec.—*Memorias do Instituto Oswaldo Cruz*, 1913. Vol. 5. No. 1, pp. 5-15. With 3 plates.
- . Citologia e Ciclo evolutivo da *Chagasella alydi*, Novo Coccidio parasito dum Hemiptero do Genero *Alydus*.—Zytologie und Entwicklungszklus der *Chagasella alydi*, einer neuen Koksidienart aus einer Wanze vom Genus *Alydus*.—*Memorias do Instituto Oswaldo Cruz*, 1913. Vol. 5. No. 1, pp. 32-44. With 2 plates.

- MESNIL (F.), CHATTON (E.), & PÉRARD (Ch.). Recherches sur la Toxicité d'Extraits de Sarcosporidies et d'autres Sporozoaires. — *Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, pp. 175-178.
- & SARRAILHÉ (A.). Toxoplasmose expérimentale de la Souris: Passage par les Muqueuses; Conservation du Virus dans le Cadavre. — *Compt. Rend. Soc. Biol.*, 1913. June 27. Vol. 74. No. 23, pp. 1325-1327.
- MOLDOVAN (J.). Beitrag zur Entwicklung des *Leucocytozoon Ziemanni* (Laveran). — *Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. Sept. 27. Vol. 71. No. 1, pp. 68-69. With 1 plate.
- NICOLLE (Ch.) & CONOR (Marthe). La Toxoplasmose du Gondi. Maladie Naturelle, Maladie Expérimentale. — *Arch. Inst. Pasteur Tunis*, 1913. Nos. 1/2, pp. 106-115.
- O'FARRELL (W. R.). Preliminary Note on a New Flagellate *Crithidia Hyalommae*, sp. nov., found in the Tick *Hyalomma Aegyptium* (Linnaeus 1785). — *Jl. Trop. Med. & Hyg.*, 1913. Aug. 15. Vol. 16. No. 16, pp. 245-246.
- PHISALIX (Mme.). Formes de Multiplication d'*Haemogregarina roulei*, chez *Lachesis alternatus*. — *Compt. Rend. Soc. Biol.*, 1913. Aug. 1. Vol. 75. No. 28, pp. 194-196.
- . Essai d'Infection sur la Vipère Aspic et les Couleuvres tropidonotes avec *Hemogregarina roulei*. — *Compt. Rend. Soc. Biol.*, 1913. July 25. Vol. 75. No. 27, pp. 110-111.
- POCHE (Franz). Das System der Protozoa. — *Arch. f. Protistenkunde*, 1913. Sept. 12. Vol. 30. No. 3, pp. 125-321. With 1 text-fig.
- v. PROWAZEK (S.). Zur Parasitologie von Westafrika. — *Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. July 29. Vol. 70. Nos. 1/2, pp. 32-36. With 1 coloured plate.
- DA ROCHA-LIMA (H.). Zur Demonstration über Chlamydozoen. — *Verhandlungen der Deutschen Pathologischen Gesellschaft*, 1913. pp. 198-210. With 10 text-figs.
- SCHILLING-TORGAU (V.). Ueber die feinere Morphologie der Kurloff-Körper und ihre Aehnlichkeit mit Chlamydozoen-Einschlüssen. II. Mit einem Zusatz über Rosssche Einschlüsse bei Syphilis. — *Centralbl. f. Bakt.*, 1. Abt., Orig., 1913. June 21. Vol. 69. Nos. 5/6. pp. 412-434. With 2 coloured plates and 1 text-fig.
- . Berichtigung zu der Arbeit Flu "Over de z.g.n. Kurloffichamen u.s.w." (Diese Zeitschr. Bd. 52. 6.) — *Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 2, pp. 351-358.
- . Zu den Bemerkungen Flu's Dl. 53, p. 359 d. Zeitschrift betreffend meine Berichtigung seiner Arbeit über Kurloffkörper. — *Geneesk. Tijdschr. v. Nederl.-Indië*, 1913. Vol. 53. No. 4, p. 604.
- SPLENDORE (A.). Nuove Osservazioni sul *Toxoplasma cuniculi*. — *Rendiconti d. R. Accad. dei Lincei.*, 1913. May 18. Vol. 22. Ser. 5. 1 sem. No. 10, pp. 722-727. With 1 plate.

See also Amoebiasis and Sleeping Sickness.



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